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BRAIN.

APRIL, 1888.

Original Articles.

EXPERIMENTS ON THE ELECTRICAL EXCITATION OF THE VISUAL AREA OF THE CEREBRAL CORTEX IN THE MONKEY.

BY E. A. SCHÄFER, F.R.S.

Jodrell Professor of Physiology in University College, London.

THE visual area of the cerebral cortex in the monkey, so far as it is determinable from the results of extirpation and of electrical excitation, comprises the whole of the occipital lobe. It perhaps includes a part or the whole of the angular gyrus, although the results of extirpation would appear to show, that the part played by the latter convolution is comparatively small. Removal of both occipital lobes produces total and permanent blindness, whereas destruction of the cortex of both angular gyri is not followed by any appreciable permanent defect of vision.[1] But, as will presently appear, electrical excitation of the posterior limb of the angular gyrus produces movements of the eyes similar to those which are produced by excitation of the adjoining portion of the occipital lobe; it is probably correct, therefore, to include at least this portion of the angular gyrus in the visual area.[1]

---

[1] H. Munk, 'Verhandlungen der Berliner physiol. Gesellschaft,' 1878; and Schafer, 'BRAIN,' January 1888. Of two monkeys in which, in conjunction with Dr. Sanger Brown, I completely removed both occipital lobes, leaving the angular gyri intact, one was totally blind and the other nearly so, having possession, however, of a small part of the visual field, so as to be able to see objects held well above the horizontal visual plane. On post-mortem examination it proved that the lesion did not in this case involve quite as much of the under-surface of the hemispheres as in the other. This agrees with the results of the excitation-experiments herein to be described, from which it will appear that stimulation of the under-surface of the posterior part of the hemispheres produces an upward inclination of the visual axes.

The results which previous observers have obtained from excitation of the visual area as thus defined are contradictory. Ferrier obtained movements of the eyes to the opposite side with closure of the lid-openings, and contraction of the pupils, from excitation of the whole of the angular gyrus, the eyes being usually directed upwards when the electrodes were applied to the anterior limb, downwards when applied to the posterior limb. He "never obtained any reactions on stimulation of the occipital lobes."[2] Luciani and Tamburini got only the conjugate deviation to the opposite side without any constant upward or downward inclination, and they also obtained similar movements, but of a less marked character, on excitation of the convexity of the occipital lobe.[3]

I have carefully explored these parts of the hemisphere with the induced interrupted current in a number of monkeys, and have obtained the following results:[4]

---

[1] A similar result is obtained on stimulation of the upper end of the superior temporal gyrus, and of the adjacent portion of the middle temporal. This may be due, as is usually supposed, to the calling up of subjective auditory sensations; but the possibility of this part also belonging to the visual area, with which it is anatomically continuous, must not be overlooked.

Whilst engaged in enumerating the parts, excitation of which produces movements of the eyeballs, it is right that the frontal area for movements of the head and eyes should be included in the list. But this is evidently different (more direct) in its mode of action, for its removal causes paralysis of those movements (Ferrier), and moreover, as I have elsewhere shown ('Proc. Roy. Soc.,' 1888, and 'International Journal of Anatomy and Physiology,' 1888), its period of latency is much shorter than that of the posterior (visual and auditory ?) areas.

[2] Ferrier, 'Functions of the Brain,' second edition, p. 244.

[3] Luciani and Tamburini, 'Sulle funzioni del cervello. Centri psico-sensori corticali.' Reggio-Emilia, 1879. (Abstract in 'BRAIN,' Vol. II.)

[4] For investigating the external surface, platinum-pointed electrodes of the ordinary pattern used in physiological experiments were employed; but for the exploration of the mesial and under surface, electrodes of a special pattern were constructed. These had their wires guarded, except on one side near the extremity, by paraffined paper, and were of such a form that they could be easily slipped between the brain and dura mater, and the excitation conducted through the unguarded parts of the wire directly to that part of the concealed surface which it was desired to stimulate.

A Du Bois-Reymond coil, with vibrating hammer, worked by a Daniell cell and with Helmholtz's side-wire inserted in the primary circuit was used throughout. Ether was employed as an anæsthetic.

Excitation of the posterior limb of the angular gyrus, the upper end of the middle temporal gyrus (into which it is continued), and of the whole of the cortex of the occipital lobe, including its mesial and under-surfaces, produces conjugate deviation of the eyes to the opposite side. The excitable area also includes the part of the mesial surface immediately in front of the internal parieto-occipital fissure (quadrate lobule). The movement produced is either a purely lateral one, or a lateral movement combined with either a downward or an upward deviation, according to the part to which the electrodes are applied.

1. When they are placed upon an area which includes the posterior limb of the angular gyrus, the part of the second temporal gyrus with which it is immediately continuous, the portion of the external surface of the occipital lobe immediately behind the external parieto-occipital fissure, and the anterior and upper part of the mesial surface immediately in front of (quadrate lobule) and immediately behind the internal parieto-occipital fissure, the deviation is accompanied by a downward inclination, which is relatively most marked when the electrodes are applied on or near the mesial surface.

2. When they are placed upon an area which includes the posterior extremity of the lobe, its tentorial surface, and the posterior and lowermost part of its mesial surface, the deviation is accompanied by an upward inclination, which is also relatively most marked on applying the stimulation on or near the mesial surface.

3. When the electrodes are applied to an intermediate area, which is chiefly formed by the remainder of the convex external surface of the lobe, but also extends over the inner border to the mesial surface, where it becomes greatly narrowed, the deviation is simply lateral.

The results, therefore, of electrical excitation of this posterior (or visual) region of the hemisphere indicate a division into three zones; not sharply marked off from, but merging into one another. The first, or uppermost zone, is connected with downward inclination of the eyes; the second, or lowermost, with upward inclination; and the third, or intermediate zone, which is wide externally and narrow mesially, with a simple lateral movement. But a lateral movement is also produced along with the downward or upward inclination by excitation of the upper and lower zones, although the vertical movement of the globes may be so powerful as almost to obscure the lateral inclination. This most frequently happens when the excitation is applied to the mesial parts of the upper and lower zones. Usually the upward direction of the visual axes, consequent upon stimulation of the lower zone, is accompanied by elevation of the upper eyelid, and the downward direction of the visual axes, consequent upon stimulation of the upper zone, by depression of the upper eyelid; but these effects have not been constant in my experiments, nor have the changes in the pupils which are sometimes observed accompanying the movements of the eyes.

I have not obtained any effect from stimulating, *with weak faradization*, the posterior limb of the angular gyrus, even when well-marked results have been obtained, with a strength of current barely perceptible to the tongue, from the parts which have just been considered.

I am unable to say exactly how far forward on the undersurface of the hemisphere these effects of excitation of the cortex may extend. The conjugate deviation to the opposite side, combined with upward inclination, is produced certainly as far as what is anatomically described as the limit of the occipital lobe on the under surface, and indeed the excitable region appeared to extend in advance of this limit; but it was not easy to say in experimenting upon this (concealed) part, to what spot precisely the electrodes had been applied.

I have occasionally obtained from stimulation on or near the quadrate lobule marked contraction of the pupils, such as is produced when a strong light is thrown directly into the eye. More usually, however, the movements, whatever the part stimulated, are accompanied by dilatation of the pupil.

So far as I have been able to observe, the visual axes always maintain parallelism when one side only of the hemisphere is stimulated. On one occasion I stimulated various corresponding points of the two hemispheres simultaneously. The result was usually a struggle between the muscles producing lateral movement, and the eyes either quivered without moving to one side or the other, or the one set of muscles preponderated over the other, so that a lateral movement to one side resulted. In one case, when the electrodes were placed on corresponding points of the mesial surfaces, there was slight convergence of the visual axes; but this result was not sufficiently marked or constant to permit much stress to be laid upon it.

If we assume that these various movements of the eyeballs are the result or concomitants of subjective visual sensations set up by the excitation, and the movements take the directions towards which those sensations are projected, then the above experiments indicate a certain connection between the parts of the cerebral visual area and of the retinæ. This connection may be stated as follows:—

1. The whole of the visual area of one hemisphere is connected with the corresponding lateral half of both retinæ.

2. The upper zone of the visual area of one hemisphere is connected with the upper part of the corresponding lateral half of both retinæ.

The lower zone of the visual area is connected with the lower part of the corresponding lateral half of both retinæ.

4. The intermediate zone of the visual area is connected with the middle part of the corresponding lateral half of both retinæ.

We may represent these relations diagrammatically, by supposing the visual areas of both hemispheres to be united at the great longitudinal fissure, and imagining each retina to be expanded and projected in its natural position upon the united area. If this is done, it is at once seen that the identical points of the two retinæ correspond with one and the same spot of the cerebral cortex; that the upper half of each retina falls on the upper part of the united area, and the lower half of each retina on the lower part of that area; that the inner half of the one retina and the outer half of the other each fall upon the same hemisphere, and that a vertical zone, including the centres of both retinæ, corresponds with the mesial part of the united area. This would connect the parts of the retinæ which are concerned in direct or central vision with a part of the mesial surface of the occipital lobes, probably including a portion of the quadrate lobules. This scheme of connection between the retinæ and occipital lobes differs in several points from the well-known scheme formulated by H. Munk,[1] as the result of extirpation experiments, chiefly upon dogs, but partly upon monkeys. The most important differences relate to the extent of the visual area, and to the part of the occipital cortex which is connected with the central parts of the retina (macula lutea). The visual area (*Sehsphäre*) of Munk is *confined to* the occipital lobes, and the maculæ luteæ are *connected with the middle of the convexity of those lobes.*[2] Consequently, if the middle of the convexity of both occipital lobes is removed, central vision should be abolished. But, contrary to Munk, I have not found this to be the case in monkeys. Indeed, it was the smallness of the effect produced by this bilateral lesion which first led me to doubt the correctness of Munk's scheme. On the other hand, in one experiment which I have performed since obtaining the above results from stimulation of the cortex, there was a marked defect of central vision produced by a bilateral injury of both mesial surfaces. Complete and permanent abolition of central vision did not result, but it was found, post-mortem, that the lesion did not involve as much of the mesial surface of the visual area, especially upon one side, as was intended at the operation. It will be necessary to follow out the question more closely by the extirpation method, and this I intend in the near future to attempt; but the difficulty of arriving at reliable conclusions from localised extirpations of the visual area is enormous. For the animals soon acquire the habit of compensating for local defects in the visual field, by rapid movements of the eyes, so as to baffle all attempts to determine the existence of such defects. I believe, indeed, that to arrive at detailed conclusions we must await the results of perimetric observations in cases of cerebral lesion in the human subject, and that these experiments upon the visual area of the monkey are chiefly of value as affording indications of the kind of result which is to be looked for in man.

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[1] H. Munk, 'Ueber die Functionen des Grosshirns,' Gesammelte Mittheilungen, Berlin. 1881, 5te Mittheilung.

[2] Op. cit. p. 127.

SCHÄFER ON THE TEMPORAL AND OCCIPITAL LOBES.

BY DAVID FERRIER, M.D., F.R.S.

THE opponents of cerebral localisation—a race well-nigh extinct—make capital out of the fact, that the advocates of this doctrine are not agreed among themselves as to the exact extent of the areas or regions supposed to be endowed with specific functions. They will therefore welcome with satisfaction the contribution to the physiology of the cerebral hemispheres, by Professor Schäfer, in the last issue of this Journal.[1]

In this paper, Professor Schäfer not only differs in important respects from all who have preceded him in this field of enquiry, but exhibits the curious spectacle of one who believes in the rigid localisation of a visual sphere, a centre for common or tactile sensibility, and differentiated motor centres, and yet contends that the important senses of hearing, taste, and smell have, apparently, no local habitation in the brain. For he can obtain no evidence of impairment even, not to mention abolition, of any of these faculties on destruction of any portion of the cortex. And in particular, he asserts, that the temporal lobe—in which it is now very commonly believed that these faculties are centralised, and where alone it would seem they can be, if not diffused among regions already otherwise occupied—may be completely removed on one or both sides, leaving hearing, taste, and smell absolutely intact.

While he thus abounds in negations, Professor Schäfer, who has done some good work in cerebral localisation, gives us no clue to the situation of the centres whose supposed seat he so ruthlessly demolishes; and he is unable to indicate any use of the temporal lobe, except that its removal on both sides is in some way the cause of a temporary idiocy.

---

[1] "Experiments on Special Sense Localisations in the Cortex Cerebri of the Monkey."—'BRAIN,' Jan. 1888.

To one who has accepted the principle of localisation in respect to vision, tactile sensibility, and special motor faculties, this should appear a most lame and impotent conclusion.

It is difficult to believe, that Professor Schäfer can have formulated to himself the logical consequences of the position he has taken up, without having had serious mistrust raised in his own mind as to the completeness of his lesions, or the reliability of his methods and observations. But if he has, his faith in his methods must be of an extremely robust character, if it can have impelled him to assume such an equivocal position, and oppose himself to the combined evidence of comparative anatomy, clinical pathology, and actual experimental demonstration.

As I have, in my work on 'The Functions of the Brain,' (page 310) attributed to Professor Schäfer (and his former colleague, Professor Horsley) opinions respecting the auditory centre altogether at variance with those which he now maintains, it is necessary that I should, in the first place, endeavour to clear myself of the charge of having misrepresented him.[1]

In a letter dated March 19th, 1885, Professor Schäfer wrote to me as follows: "We have some monkeys just now which you ought to see. The whole of the temporo-sphenoidal lobe, including the hippocampus, has been removed in them, and we cannot detect any paresis of sensation (except auditory)."

I did not, however, doubt the accuracy of my own observations, and, at my request, Professor Schäfer and his colleague consented to perform the experiment under my supervision and direction. When this was carried out, I had no difficulty in demonstrating to their satisfaction that the lesion had caused profound impairment of common and tactile sensibility on the opposite side of the body. And I had the further pleasure of suggesting to them, and seeing performed, experiments which I had myself planned, viz. the extension of the lesions into the gyrus fornicatus, or anterior division of the limbic lobe. The results of these experiments demonstrated the physiological unity of the callosal and hippocampal gyri, and confirmed the unity which Broca had indicated on anatomical grounds.

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[1] A charge which I observe is made in the 'Phil. Trans.,' 1888, B. 20, p. 20, footnote.

I naturally gathered from Professor Schäfer's letter that he had obtained evidence of the existence of an auditory centre in the temporal lobe. For he expressly excepted auditory from the negative effects as regards other forms of sensibility.

And as he had informed me that—in harmony with my own experiments—he had obtained no indications of impairment of hearing on destruction of the rest of the temporal lobe, apart from the superior convolution, I considered myself justified in quoting his results as confirmatory of my own. Professor Schäfer is of course entitled—nay bound—to change his views, when the evidence on which he based them appears on further consideration to be insufficient; but I do not think that I can be said to have seriously misrepresented them as they at that time stood.

Professors Schäfer and Horsley had given me an opportunity of testing, and I am happy to say of correcting, conclusions which they were inclined to draw adverse to my own. But the first intimation I had of Professor Schäfer's further investigations (in conjunction with Dr. Brown) was the announcement at a meeting of the Physiological Society on February 12th, 1887, of the views which he had already then arrived at, and now puts forward in a more elaborate form. These were first published in the 'Proceedings of the Physiological Society,' No. 2, 1887. I criticised them at the time, and at a subsequent meeting of the Neurological Society, I strongly deprecated this premature publication of conclusions while the evidence was incomplete—seeing that the animals were still alive, and the actual lesions undetermined—as being likely to do harm and prejudice dispassionate enquiry. And I particularly called attention to—and here emphasise—the difficulties that have to be encountered, and the fallacies that are apt to be committed, in investigating the sense of hearing in the lower animals. Care must be taken to discriminate between the sense of hearing proper, and mere reflex reaction to sudden, sharp, or loud sounds, a reaction not incompatible with real deafness. Mere coincidences in animals keenly on the alert to everything going on around them, must be scrupulously eliminated. The tests of hearing should be such as to entirely avoid attracting the animal's attention by other channels of perception, such as sight, imitation of other animals, a sense of proximity conditioned by vibration, agitation of the air, and such like. The animal's present behaviour should be compared with its past, or with that of normal monkeys under conditions of variation in sound, and special kinds of sound, only, all other things remaining the same. And even then, it often requires long and laborious investigation to eliminate all sources of fallacy. Professor Schäfer is evidently satisfied that he has avoided all sources of fallacy himself, and he quotes in support of his conclusions the opinions of a Committee of the Neurological Society in favour of the retention of hearing in two of his experimental animals, in one of which he says he had scooped out the superior temporal convolution on both sides, "so that not a trace of the convolution in question should remain" (Fig. 5); and in the other he had removed not only the superior temporal convolution, but even the whole temporal lobe on both sides (p. 373).[1]

But Professor Schäfer's figures (Figs. 5 and 6) of the brains of these monkeys must be regarded more as a sort of pictorial representation of his own idea, or wish, as to the extent of the lesions, rather than of the reality. Having myself examined both preparations, I most emphatically deny the accuracy of Professor Schäfer's diagrams. In neither the one nor the other were the superior temporal gyri completely destroyed:—considerable portions remaining absolutely intact in both hemispheres, and notably so in the brain which Fig. 6 professes to represent. And considering how extensive lesions may be in any given centre—witness the visual centre—without total annihilation of its functions, nothing short of absolute destruction, primarily or secondarily, is likely to abolish all vestiges of the sense to which it is related.

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[1] Professor Schäfer has committed a slight irregularity in quoting the opinions of a Committee, before the members of the said Committee had met together, or formulated any report of their proceedings.

In the absence of any information as to methods and tests employed by the visitors appointed by the Society, I am willing, without criticism, to attach the utmost weight to the opinions at which they arrived. But of this I have no doubt whatever, that if the evidence of hearing in the animals submitted to their investigation was indubitable, the explanation is to be found in the fact, that the superior temporal gyri were not completely removed in any one of them.

But apart from Professor Schäfer's experiments, let us proceed to consider the question of the locality of the sense of hearing on broader grounds. The evidences of the localisation of the centre of hearing in the temporal lobe—and according to my view, in the superior convolution of this lobe—are as follows: First, the reaction on excitation by the electrical stimulus—a reaction which afforded me the suggestive indication, leading to the verification by destructive lesions, of the relation between this convolution and the faculty of hearing. Electrical irritation of the upper two-thirds of the superior temporal convolution causes a quick retraction or pricking of the opposite ear, as if towards the supposed origin of the sound, combined generally with turning of the head and eyes to that side, and dilatation of the pupils. I have used the term "pricking" as synonymous with retraction and pointing of the auricle in the direction to which sound would naturally be referred. I have nowhere described the reaction as a "pricking *forward* of the ear," such as Professor Schäfer attributes to me (p. 373). The reaction is precisely that which an animal makes when a shrill sound is made towards one side, as may be easily verified in any monkey, or such animal as the cat, whose ears, when at rest, are pointed and directed forward. In the lop-eared rabbit, however, irritation of the homologous region causes sudden elevation of the ear, as well as retraction and exposure of the mouth of the auricle towards the referred origin of the sound. Professor Schafer says that excitation of very various parts of the cortex (frontal region, middle temporal gyrus, angular gyrus, and occipital lobe) produces "almost precisely the same effects" (p. 372). As I was myself the first to point out the various positions from which movements of the head and eyes to the opposite side might be stimulated, it is scarcely likely that I should describe the movements of the head and eyes as a reaction peculiar to the superior temporal convolution. The special feature of this reaction is the sudden retraction of the ear which occurs on irritation here, and here only. Professor Schäfer's ingenuousness is not very apparent when he endeavours to conceal the significant differences between the reaction of the superior temporal convolution and that of the other regions above enumerated, under the words "*almost* precisely the same effects."

Next, as regards the evidence from destructive lesions, as supplied by my own experiments, and those which I made in conjunction with Professor Yeo. This consists of an extensive series of experiments reported in detail in the 'Philosophical Transactions,' Part 2, 1875, and Part 2, 1884, involving divers lesions of the temporal lobes;—*negative* as regards affections of hearing when the superior temporal convolution was intact, and *positive* when this was destroyed unilaterally and bilaterally; and all mutually corroborative of each other. No greater importance is attached to these experiments individually than as links in a chain leading up to the conclusive demonstration which was given before the Physiological section of the International Medical Congress in 1881.

In one of the series in which the superior temporal convolution was destroyed on both sides in the upper two-thirds of its extent (the left angular gyrus having been destroyed previously but without permanent effects), the following notes were made as to the condition of the animal on the day after the operation.[1]

> "*April 8th*, 10 A.M.—Animal alive and active, and jumped out of its cage when the door was open. Sight was good, and tactile sensation unimpaired. Various experiments were made to ascertain the existence or not of hearing; but it was difficult to devise a test, as the animal was continually on the alert; and it was not easy to make a sound without in any way attracting its attention by sight. The following method was tried: While the animal was sitting quietly by the fire, I retired to the other room, and while watching through the chink of the half-shut door, called loudly, whistled, knocked on the door, tinkled glass, &c., without ever causing it to look round, or give any sign of having heard. I then cautiously approached the animal, and not till it saw me did

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[1] See Experiment 15, 'Phil. Trans.,' vol. 165, part 2, 1875.

it give any sign of consciousness of my presence. When the same experiment was repeated, while the monkey and its companion were quietly seated by the fire enjoying the heat, it gave no sign of hearing; while its companion started with alarm, and came with curiosity to ascertain the cause of the sound. . . . 8 P.M.—In the presence of Dr. Burdon Sanderson I repeated the various tests with the view of eliciting signs of hearing. To all it remained without response. It seemed unconscious of my presence when speaking close to its ear, and only started when it caught sight of me."

The last of the series confirming the presence of, and establishing the permanency of, similar symptoms was the now somewhat famous monkey exhibited at the International Medical Congress. In view of certain questions that have been raised as to the exact condition of this animal, I give here verbatim the rough notes written down from day to day.

The subject of the experiment was an unusually active and intelligent monkey, in full possession of all its faculties.

It was designated Monkey F. The extent of the lesions, superficial and deep, in this animal are faithfully represented by photography on Plate 22, Figures 23–38.[1]

"*June* 23*rd* (24 hours after the operation).—F. to-day is rather dull and drowsy-looking. Some œdema of face and eyelids. Right eye partially closed, the left open. Put out its hand and touched companion sitting near it and eating a piece of bread. It gives distinct signs of perception of touch on hands, feet, ears; moves about perfectly freely. Pays no attention whatever to the loudest sounds which startle the other monkeys, or to whistling, tapping, &c., close to its ears. Oil of bergamot placed on a rod and held at its nostrils caused it to sniff apparently and make movements with its lips. Acetic acid causes it to retract its head sharply, and sneeze or snuff out. Acetic acid to lips causes licking movements of tongue; so also common salt placed on lips. Sees perfectly well, and goes about from one cage to the other through small door.

"*June* 25*th*.—F. is very bright and active; runs about actively climbs up bars of cage, and peers eagerly through bars at what is going on. Takes things offered it. Took a piece of date, smelt it and threw it down; so also a piece of bread. Took next a piece of cold boiled potato, smelt it, rubbed it in its hands, and began to eat with relish. Pays no attention to sounds—tapping, scratching, &c.—on side of cage, which alarm its companion toward either side. This was carried on repeatedly for the space of an hour without eliciting any sign of perception. Going down to the place where the animals are kept, sets all the others on the *qui vive.* No sign of this on the part of F. But as soon as I come within sight, it looks interested and comes to the bars and looks up.

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[1] 'Phil. Trans,' p. 2, 1884.

"*June 27th.*—Examined F. with Dr. Lauder Brunton. Tried various tests, sounds, whistling, &c. Could not decide positively. Animal occasionally turned its head, but whether coincidence or hearing, doubtful. At any rate, conclusion arrived at that if the animal not absolutely deaf, its hearing was profoundly affected. Noted that the ears did not twitch as in the case of the companion monkey; nor did it show alarm at sounds.

"*June 28th.*—F. placed in a separate compartment. Did not reply to plaintive cries of its companion. F. tried constantly to see its companion through small apertures in cage. Loud sounds, which made all the others start and look frightened, caused generally not the slightest sign on the part of F. Occasionally it turned its head coincidently with the noise, but whether hearing, very problematical, as at other times no such sign. The ears did not twitch; nor any nervous wincing as in the case of the companion. No response to cries or calling by name. Sight, motor power, and tactile sensibility perfect to all tests.

"*June 30th.*—Repeated the tests with the same result as formerly. The animal is mostly totally unconcerned when its companion is in abject terror, shrinking, winking, and twitching its ears. Every now and then, doubt seems raised by the animal looking at the same time as a noise is made.

"*July 2nd.*—Again examined the animal carefully with Dr. Yeo. In response to various tests—sounds of various kinds—there was most commonly no indication of perception, such as the companion monkey showed by shrinking, grimaces of terror, cries, and twitching of the ears. Occasionally the animal would turn its head coincidently with the sound. But this is only if a sound made towards stairs leading down to cage, where it in general directed its attention spontaneously. *Conclusion:* if not absolutely deaf, hearing very doubtful, and at least no impairment of the other powers of the animal.

"*July 5th.*—Made several experiments with electric bell placed against side of cage. On ringing it, the companion was at first very anxious about the sound, while F. paid no attention, but after a time it ceased to regard it. Cannot yet be sure whether the animal hears at all, but certainly is not at all disturbed by sounds which make the others start, twitch ears, wink, &c. No twitching of the ear in F. Otherwise perfectly well. Runs about; is very expert in catching flies, which it watches carefully and pursues skilfully. Very much on the alert to all that goes on, and occasionally utters a grunt if it sees anything like water, food, &c., which it wants. Never, however, replies by grunts to those of its companions, and is generally perfectly silent. Formerly it was continually in the habit of grunting, &c., in reply to name, &c.

"*July 7th.*—Have been making numerous experiments, also Dr. Yeo, as to whether the animal hears the noise of a percussion-cap exploded near. It is most difficult to judge. Occasionally it seems to start coincidently with the sound, and yet at other times gives not the slightest sign, while the others invariably start. The dressings removed to-day; all aseptic; wound perfectly healed. Animal well and vigorous, very much on the alert. The ears are now mobile. It moves them during its actions of attention, but I have not yet been able to ascertain definitely whether they twitch to sound. This afternoon, at six, made an interesting experiment while sitting in the cage with its companion (G.), and both quite still, and engaged in examining each other. I made repeatedly a scraping sound on floor with foot as if going up to cage. The companion G. invariably opened its eyes and looked up, whereas there was never any sign on the part of F.

"*July 13th.*—Examination to-day confirms former remarks. Once very clearly percussion-cap excited G., and met with no sign of response from F. Does not answer calls like G., and pays no attention to sounds which cause G. to look up.

"*July 15th.*—Well and active. Gives no signs of recognition to cries, and frequently not the slightest sign of movement, or attention to shots. It is more than doubtful whether there is any reaction at all.

"*July 20th.*—Condition unchanged.

"*July 27th.*—Owing to the death of G., F., which has been disconsolate, and occasionally uttering plaintive cries, was transferred yesterday to the cage of C. (hemiplegic). The two seemed to agree tolerably well, with the exception of squabbles as to food—C. being very indignant, and occasionally attacking F. for seizing the food first. Made various experiments as to hearing, and it seems that the signs of reaction (which have caused so much doubt) to sounds are less evident. The electric bell was transferred to the side of its cage. Ringing it suddenly, caused C. to be very much disturbed and annoyed, whereas F. paid no attention whatever. Coming downstairs to the place where the animals are kept, I observed C. looking out attentively, but F.

remained in back of cage, not coming forward until such signs on the part of C. as to attract notice. Repeated percussion-cap experiments several times. C. invariably started, but to-day I failed to detect any unequivocal indication of this on the part of F. As to other sounds—calling, whistling, &c.—F. pays no attention whatever, whereas the attention of the other monkeys is at once excited. The animal is excessively on the alert, and it follows every movement, and is with difficulty surprised.

"*July* 29*th.*—The percussion-cap experiment, repeated to-day several times in the presence of Dr. Yeo and J. G. Romanes, was every time negative, causing no reaction or starting, while its companion was invariably much startled and annoyed.

"*August* 4*th.*—The animal was exhibited to-day before the Physiological section of the International Medical Congress. A percussion-cap was exploded close to it several times before the audience, and it gave no sign whatever of starting; whereas C. started very emphatically.

"*December* 12*th.*—The animal has kept in excellent health, and is most lively, and continually engaged in active gymnastics. Many experiments were made with the view of testing its hearing. Sometimes it appears to start when a pistol-cap is exploded, so that occasionally it is judged to hear. To-day tried the experiment of calling from the top of stairs. The other monkeys [2] are both on the alert, looking eagerly and making sounds of expectation; but F. pays no attention whatever, and goes on with its own occupation quite unheeding. It does not pay any attention to scratching on cage as the others do, and in general does not respond to sound-calls, &c., which always excite the attention of the others. It does not utter sounds in reply to calls. Occasionally, when left all alone, it utters a short cry of loneliness.

"*March* 31*st.*—While sitting to-day having its back scratched, a pistol-cap was exploded close to cage; not the slightest start, though the others and Mr. N., who was scratching the monkey, made a sudden start.

"*July* 27*th.*—The animal has remained well and lively without showing any new symptom, and still to all appearance exactly in the same condition as before as regards hearing. To-day, made another series of observations of the animal while in its cage, and also after it was removed and placed in a small cage on table. Loud whistling, knocking, ringing electric bells in the neighbourhood failed to elicit any sign of attention. Occasionally, as before, there was an appearance of a start, or a look round, apparently conditioned by the noise, but repeated experimentation showed that this was pure coincidence."

I was not a little amused at listening to Professor Schäfer's comments on the above record which I read before the Neurological Society. First, he suggested that the notes themselves showed that the animal was not really deaf. And this, I admit, was in some respects an intelligible and fairly defensible position to assume. For doubts were constantly being raised, as the record freely states, in my own mind as well as in that of other observers.

It was only after long and repeated investigation, and variation of tests, that our doubts as to the animal's true deafness seemed fairly dispelled.

The only reaction which might be interpreted as a sign of some obscure sense of hearing was the occasional start coincidently with a loud or shrill sound made in its neighbourhood. But even granting that this was not mere coincidence, it might be due to some remnant of the centre, or co-exist with abolition of true auditory perception, and resemble the reaction to loud sounds which may be elicited in animals entirely deprived of their cerebral hemispheres, as Flourens and Longet demonstrated long ago.

But almost in the same breath Professor Schäfer next suggested that we had in truth lighted on a specimen of a monkey stone-deaf before the operation. It has been said that a dead donkey is difficult to find. A deaf monkey will be a greater curiosity. And when Professor Schäfer succeeds in finding one, it will not help him in his argument, as this animal's hearing powers were, as the record shows, well established before it was submitted to operation.

Professor Schäfer does not in his paper expressly repeat his brilliant inspiration, but, with a generosity which I do not fail to appreciate, he sums up his estimate of the evidence which Professor Yeo and I have given as to the localisation of the auditory centre, with the declaration of his belief, that he is "justified in asserting that the supposed localisation of the auditory perceptive faculties in the temporal lobe in monkeys, has no experimental evidence in its favour" (p. 377). Without disputing the accuracy of the views of Munk and Luciani, who have satisfied themselves that lesions of the temporal lobes in dogs cause total abolition, or profound impairment, of the sense of hearing, he apparently takes comfort from the fact, that these physiologists have not actually experimented on monkeys. I am not concerned to speak for Munk or Luciani—believing, as I do, that they are both wrong as to the exact definition of the auditory centre in the temporal lobe—but they themselves, and doubtless all who believe in comparative anatomy and physiology, have no hesitation in extending their conclusions to monkeys and man. For if the localisation of an auditory centre is true of dogs, it must *à fortiori* be true of higher animals, in which cerebral localisation is more conspicuously manifest.

If, however, it should still appear that the results of experiments on the lower animals are not absolutely conclusive, let us turn to the records of human pathology, which is, after all, the *ultima ratio* in questions relating to subjective conditions, not clearly indicated by external manifestations. Deafness from cerebral disease is undoubtedly a rare occurrence. That this should ensue, requires the existence—rarely happening—of symmetrical lesions in both hemispheres; for unilateral lesions of the auditory centre do not appear to cause, at least permanent, affections of hearing. But as destruction of the motor centres of articulation in the left hemisphere causes aphasia, so destruction of the auditory centre on the left side causes the now universally recognised condition known as "word-deafness," a condition alone explicable on the hypothesis of the existence of an auditory centre. In every instance of this kind which has been examined post-mortem, the destructive lesions have involved the temporal lobe, and in all, the superior temporal convolution.

I have been able to find only two uncomplicated cases of symmetrical lesion of this region in both hemispheres, but both are of transcendent importance. Shaw[1] has recorded the case of a woman, ætat. 34, who, two months before her admission into his asylum, lost power in the right arm, and soon after had a sudden apoplectic seizure resulting in loss of speech and deafness. The loss of power in the right hand soon passed off. She became incoherent, more or less maniacal, and demented. On admission, she was found to be perfectly *deaf and blind.* She died of pneumonia a year afterwards.

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[1] 'Archives of Medicine,' Feb. 1882.—Abstract in 'BRAIN,' Vol. V., 1882–83, p. 430.

Post-mortem examination showed *complete atrophy of the angular gyri and superior temporo-sphenoidal convolutions of both hemispheres* (see figures accompanying the paper). The grey matter of the atrophied regions had entirely disappeared, leaving the outer layer attached to the pia mater, with a cavity underneath formed at the expense of the grey matter.

The cranial nerves were normal in appearance, the optic nerves showed increase of the connective tissue septa, atrophy of the nerve-fibres, and spaces filled with a colloid-like material.

This case may be objected to, as not being absolutely conclusive, owing to the fact, that possible disease of the organs of hearing is not expressly disproved in the record; though the sudden onset of deafness coincidently with the cerebral seizure would almost of itself negative such a hypothesis.

No such objection can be urged against the following case reported by Wernicke and Friedländer.[1] As this case is so important, I give the following particulars at some length.

A woman, aged 43, who had never suffered from deafness or affection of vision, was attacked on the 22nd of June, 1880, with right hemiplegia and aphasia. She remained in the hospital until August 4th, when she was discharged. At this time the patient could speak, but she spoke unintelligibly, and was sometimes believed to be intoxicated. She not only could not make herself understood, but she could not understand what was said to her. She was received into the hospital again on September 10th, with slight paresis of the left arm. The right hemiplegia had entirely disappeared. The patient was looked upon as insane. She was absolutely deaf, so that she could not be communicated with. She died of an attack of hæmatemesis on the 21st of October. An extensive lesion was found in each temporal lobe, invading the superior temporal convolution on both sides (see figures accompanying paper). The rest of the brain exhibited no abnormality; nor were there any conditions leading to increase of the intra-cranial pressure and possible affection of the cranial nerves. It was proved that the patient had previously enjoyed excellent hearing. Her total deafness occurred rapidly in connection with the other indications of cerebral disease. In order to exclude possible local affection of the ears, these organs were submitted to examination by Professor Lucas. The result of this examination was practically entirely negative, only a slight dry catarrh being found, and nothing locally to account for deafness.

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[1] 'Fortschritte der Medicin,' "Ein Fall von Taubheit in Folge von doppelseitiger Laesion des Schläfelappens," Band i., No. 6, March 15th, 1883.

This case will, I venture to believe, be held to justify the conclusions drawn by the authors themselves, viz. "that the auditory nerves ultimately end in the temporal lobes, and that double or bilateral lesion in these lobes causes complete deafness. It may therefore be stated with all certainty that the temporal lobes are the cerebral centres of hearing."

And I should myself go further and say, that the case is entirely in harmony with my own more strict localisation of the auditory centre in the superior convolution of this lobe.

The experimental evidence in favour of the localisation of special centres of smell and taste, which has, up to the present, been adduced by myself in the case of monkeys, and by Munk and Luciani in dogs, cannot be said to possess such precision in respect to the position and extent of these centres as not to stand in need of further investigation. My own work in this direction has been mainly that of the pioneer. But these experiments, with all their deficiencies and imperfections, taken by themselves alone, furnish at least a strong presumption, that the olfactory centre is situated in the hippocampal region of the temporal lobe, at its lower extremity. But when taken with the comparative development of the hippocampal or pyriform lobule in *osmatics* and *anosmatics*, and the anatomical relations of this region to the olfactory tract, and anterior commissure, they constitute a body of evidence in favour of the localisation of the olfactory centre mainly, if not exclusively in this situation, which I venture to think will not be considered as materially weakened by Professor Schäfer's inability to detect any impairment of the sense of smell in those animals in which he professes to have completely removed this lobe.

Investigations as to the sense of smell in the lower animals are perhaps more difficult than that of any of the other sensory faculties, seeing that the external manifestations of olfactory perception proper—apart from those caused by substances possessing some degree of pungency and not strictly pure olfactory stimuli—are not so evident as to ensure detection without a long previous study and minute acquaintance with the habits of the animal.

But inasmuch as Professor Schäfer, while unable to confirm, is also unable to adduce any facts opposed to the positive results obtained by Munk, Luciani and myself, it does not seem necessary to discuss the question at greater length. I would only remark that, if Professor Schäfer is right in his conclusions that the *whole* of the temporal lobe, including the cornu Ammonis and hippocampal lobule, may be removed without abolishing or impairing the sense of smell, he must be considered as having proved more than he could wish:—viz. that comparative anatomy is a delusion and a snare.

The exact position of the gustatory centre is admittedly less clearly defined than any other.

I have related two experiments[1] in which the lower half of both temporal lobes was extensively disorganised. In these animals no reaction was induced by placing on the tongue such substances as aloes, citric or acetic acid, which in normal monkeys excite active expressions of disgust and discomfort. I do not profess to have determined the connection between these symptoms and the destruction of any particular region more precisely than that above described; nor were the animals kept sufficiently long alive to decide the question as to their permanency, but the observations are not without important significance.

Professor Schäfer objects, that the animals "were in an entirely abnormal condition when tested, nor was the testing performed with purely sapid substances, but with acid and aromatic particles."[2] The only abnormal conditions present

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[1] Experiments 14–15, 'Phil. Trans.,' vol. 165, Part 2

[2] P. 378, footnote.

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in these animals, different from others similarly tested, were the position of the lesions:—viz. in the lower half of the temporal lobes. And even if aloes, colocynth, citric and acetic acids are not "purely sapid substances," but "also acid and aromatic particles," the objection to their use seems somewhat irrelevant, seeing that no reaction whatever was induced by any of them.

Let us now turn to Professor Schäfer's utterances in respect to the visual centre of the cortex. The chief point at issue between him and myself is the anterior boundary of the visual zone. While he considers it as being sharply bounded by the parieto-occipital fissure, I maintain that it embraces also the angular gyrus—the two constituting together what I have termed the occipito-angular region.

With respect to the effects of electrical irritation of this region, it is true that while *invariably* obtaining movements of the eyes from the anterior and posterior limb of the angular gyrus—and in this I am absolutely confirmed in every instance by Luciani and Tamburini—I did not observe similar movements, or other external manifestations, on irritation of the occipital lobes alone. But inasmuch as Luciani and Tamburini did *occasionally* observe similar movements of the eyes on irritation of the occipital lobe, though less marked than in the case of the angular gyrus, and as Professor Schäfer seems to have had no difficulty in always obtaining the same, I am not disposed to question the accuracy of their results, and will admit that I may have overlooked them, though actually present in the ten animals on which I experimented; or that the parts may have lost their usual excitability owing to some temporary conditions. But this admission only strengthens the position which I have maintained. For if the same, or similar, movements are capable of being excited from the occipital lobe, as well as from the angular gyrus, the presumption is that the regions in question have a community of function. And I am glad to observe that Professor Schäfer adopts my view, that these movements are the expression of subjective visual sensation.[1]

Professor Schäfer is unable to confirm the results obtained by myself (and in conjunction with Professor Yeo) on destruction of the angular gyrus; and he is inclined to attribute such effects as we have observed to mere functional perturbation of the true visual centre situated in the occipital lobe.

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[1] Proceedings of the Roy. Soc., March 1, 1888.

I will, for the purposes of argument—but only so far—admit that the temporary total blindness consequent on bilateral destruction of the angular gyrus, may be explained in the manner suggested by Professor Schäfer; and I will therefore exclude these experiments from consideration. But inasmuch as it is held by Professor Schäfer, Munk, and others, that unilateral destruction of the visual centre always causes homonymous hemiopia, and not crossed blindness, or amblyopia, the results of destruction of the angular gyrus, if merely attributable to functional perturbation of the occipital lobe, should in all cases assume the form of hemiopia. How then are the following facts to be explained in accordance with this hypothesis?

In one animal[1] the left angular gyrus was cauterized with the galvanic cautery.

> "The left eye was secured, and the animal allowed to recover from stupor. At the end of half an hour, it was evidently wide awake, but would not move unless touched. At this time it was removed from its cage and placed on the floor, whereupon it began to grope about in a sprawling manner, knocking its head against every obstacle. After some minutes of this behaviour, it subsided, and refused to move. It made no sign of fear at threatening gestures, and did not wink at a thrust of the finger at its eye, until the finger almost touched the conjunctiva, when the usual reflex closure occurred. Half an hour later, the same tests were employed with precisely the same indications of total loss of vision. At the end of still another half an hour, while it was lying quietly in its cage, it was gently laid hold of without noise to attract its attention, whereupon it bounded away with an expression of fear and surprise, and ran full tilt against the leg of the table, where it remained groping and sprawling for a few minutes. It then started off, and this time ran against the wall, against which it sprawled helplessly. Similar things are repeated. It gave no sign of perception when it was cautiously approached without noise. But when a slight noise was made with

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[1] Experiment 5, 'Phil Trans.,' Part 2, 1884.

the lips quite close to it, it darted off and came against the wall as before, where it lay down. Half an hour later, while it was resting quietly in a corner, with its eye open, the light of a lantern was flashed in its eye, but it gave no sign. Creeping up to it cautiously without exciting its attention, the observer made a slight whisper close to its face, whereupon it peered eagerly; but evidently remembering the results of running away, it crouched down and would not move. Half an hour later, when it was quiet in its cage, it started suddenly on being touched, and ran its head into a corner, where it crouched.

"Next day, its left eye being still closed, it showed unmistakably the possession of vision with the right eye. It laid hold of things as usual, and ran about the laboratory in every direction, passing obstacles right and left with perfect precision, and ducking its head to pass underneath bars as it ran along the top of the waterpipes of the laboratory. No defect of vision, amblyopic or hemiopic, could be detected."

In another animal,[1] the left angular gyrus was cauterised up to the parietal-occipital fissure (the posterior part of the corpus callosum being also divided at the same time).

"The left eye was securely closed, and the animal allowed to recover from its narcotic stupor. In half an hour it began to move about spontaneously, although rather unsteadily. An hour and a half after the operation it walked about the laboratory, knocking its head against legs of chairs, and other obstacles in its path. When a piece of apple was held under its nose, it grabbed it and ate. It continued to walk about here and there, every now and then coming to a dead halt full tilt against the wall. Three hours after the operation, it again, in running about the laboratory, came full tilt with its snout against the wall, where it rested. While it was resting quietly, we crept up to it; but the animal, though with eye wide open, and looking towards us, made no sign of perception. Threatening grimaces were likewise without effect; but on making a noise with our lips, the animal seemed alarmed, peered forwards, and yet, though it came quite close to our faces, seemed to see nothing. It was tried to right and left in the same way, but there was no sign of vision, to one side or the other. Next day, the left eye being still closed, the animal ran about in every direction, ducking under bars, passing objects to right and left with the utmost precision, and never once knocking against anything one side or the other. Not the slightest impairment of vision could be detected; and it was able to pick up the minutest objects lying about its cage, or thrown down near it."

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[1] Experiment 7, 'Phil. Trans.' part 2, 1884.

Similar results were obtained in many other instances, and if in some they were even more transient, this may have been due to less extensive destruction, or perhaps to differences in animals as to the relative importance of this region in the ordinary exercise of their visual faculties.

But these facts, carefully noted and faithfully recorded by Professor Yeo and myself, are altogether unintelligible on the hypothesis that they are merely due to functional perturbation of a centre, the destruction of which causes only hemiopia. If, nevertheless, it should still be asserted that the results, being so transient, indicate rather functional perturbation than the cross relationship which I hold to exist between the eye and the angular gyrus, it would be natural to expect, that the much more severe operation of actual extirpation of a large portion of both occipital lobes would produce a greater disturbance of the visual centres than the mere erosion of the surface of the angular gyrus. How then shall the following facts be explained?[1]

"The occipital lobes were exposed on both sides in a monkey, and the surface exposed destroyed by the cautery, which was also passed deeply into the interior of the lobes so as to cause as much disorganisation as possible. Care was taken not to injure the angular gyrus. The operation was completed at 3.30 P.M.

"4.10 P.M.—The animal after lying in a state of stupor until now, begins to move, but staggers a good deal. The eyes are open, and the pupils are dilated. It indicated consciousness by turning its head when called to.

"5.45 P.M.—Gives emphatic evidence of sight; ran away when I approached it, carefully avoiding obstacles. Seeing its cage door open, it entered, and mounted on its perch, carefully avoiding the cat which had taken up its quarters there. Tried to escape my hand when I offered to lay hold of it, but picked up a raisin which I had left on the perch."

Here is a case of extensive destruction of both occipital lobes, and yet in little more than *two hours* after this serious operation, the animal gave the most emphatic evidences of precise vision. Or take the facts of the following case[1] :—

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[1] Experiment 22, 'Phil. Trans.,' vol 165, Part 2.

> "The occipital lobes were exposed in a small, rather weakly monkey, and the lobes severed by a perpendicular section with hot wires about a quarter of an inch posterior to the parieto-occipital fissure, so as to avoid all interference with the angular gyrus. The operation was completed at 4.30 P.M., the animal having by the time the wound was dressed almost completely regained consciousness.
>
> "4.45 P.M.—Began to move about in rather a staggering manner, but exhibiting no muscular paralysis.
>
> "4.55.—Can see quite well, as it avoids obstacles, and when removed, regains its place by the fire."

Here is a case in which, after removal of at least two-thirds of both occipital lobes, the animal indicated its possession of vision *within half an hour* after the operation.

Or take a third case[2] :—

> "In this animal the occipital region was exposed on both sides, and *both occipital lobes* were severed with the galvanic cautery and scooped out bodily. The line of incision in both cases passed between the anterior extremity of the first occipital, and the parieto-occipital sulcus. The portion removed on the right side weighed 1·9 grammes; that removed on the left weighed 2·1 grammes [fully two-thirds of the whole].
>
> "In half an hour after the operation the animal sat up and wanted to move about, but it was kept still, wrapped in a blanket to prevent the risk of hemorrhage. Both eyes were open equally, the pupils smallish, and the conjunctival reflex equally distinct on both sides. For two hours, though it was looking about as though it saw, no definite test of vision was made; but at the end of that time, it went and sat down beside the next cage, and allowed the other monkeys to handle it and examine its head. It then, of its own accord, went into its own cage and sat down. A piece of apple was thrown into the cage, and though it fell a full arm's length away, the animal, without the slightest hesitation or want of precision, put forth its left hand, picked it up and ate. Half an hour later, similar tests were made. A piece of apple was thrown near it; this it took and began to eat: on the piece being taken

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[1] Experiment 28, 'Phil. Trans.,' vol. 165, part 2.

[2] Experiment 9, 'Phil. Trans.,' 1884.

from it, it seized another piece lying in the cage and ate this, and having finished it, came forward a few steps and picked up another piece lying on the floor."

There was thus the most complete proof of retention of vision. Here again, *within two hours* after removal of at least two-thirds of the occipital lobes on both sides (see photograph plate 21, Fig. 15) the animal indicated sufficiently clear vision to be able to pick up a minute object lying on the floor. Within, therefore, the time at which animals with unilateral or bilateral destruction of the angular gyrus are blind on one or both sides, the much more formidable operation of destruction of the greater portion of both occipital lobes leaves the animal's power of vision practically unimpaired, so far as ordinary tests are concerned. It is surely therefore unreasonable to ascribe the effects of mere erosion of the angular gyrus to functional disturbance of the occipital lobe.

I do not, however, argue—as I at first believed—that the occipital lobes do not form part of the visual centres, but conclude from these facts, that greater relative disturbances of vision occur from lesions of the angular gyri than from much more extensive lesions of the occipital lobes. They show also, what an enormous amount of destruction can be inflicted on the sensory or perceptive centres of the cortex without obvious impairment of their functions.

I have always, in contending for the negative effects of extensive removal of the occipital lobes, specially made the proviso, that the lesions shall not extend up to the parieto-occipital fissure. For I was myself the first to point out that, when the lesions extended so far forwards, impairment of vision, or even total blindness, ensued—results which I attributed to direct implication of the angular gyrus, or its medullary connections.

That this is the real explanation of the total blindness obtained by Munk and Schäfer on removal of the occipital lobes, it will not, I think, be difficult to demonstrate, and that, too, even from Professor Schäfer's own facts alone.

If his Figure 3, p. 368, be examined, which represents the extent of the lesion in a monkey said to have been rendered totally and persistently blind, it will be seen that the lesion extended on the occipito-temporal aspect beyond the anterior limit of a line drawn along the course of the parieto-occipital fissure. On both sides, and particularly on the left, the lesion extends well into the collateral fissure and beyond the anterior boundary of the occipital lobe proper. (A similar condition, even more marked, exists in the brain of the hemiopic monkey, Fig. 2, as I have found on personal examination.) If the next figure—Fig. 4—be examined, in which vision was not abolished, it will be seen that the lesion, though it extends slightly beyond the limits of the occipital lobe proper, does not extend so far forwards as in the first case.

On this the whole question turns.

Professor Schäfer admits that, with the exception of this difference, the removal of the occipital lobes was equally complete in both cases. It is obvious, however, that if it were merely a question of removal of the occipital lobes, the results should have been precisely the same. The conclusion to be drawn from this is that the total blindness was due to implication of the region lying in front of the line of the parieto-occipital fissure. Other facts, some of which have also been supplied by Professor Schäfer himself, place this beyond all doubt.

I have recorded several instances[1] in which in attempts to remove the hippocampal region, I have observed, in addition to other symptoms, hemiopia of greater or less degree and duration. In one of these[2] there was only superficial erosion of the surface of the inferior occipital convolution; but at the anterior extremity of this gyrus, where it becomes continuous with the middle temporal convolution, and just at the lower extremity of the parieto-occipital fissure, the lesion became more deep, and thence extended along the middle temporal convolution, detaching all below this.

In another case[3] the occipital lobe was altogether uninjured, the lesion commencing just below the inferior extremity of the parieto-occipital fissure.

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[1] See Experiments 27, 28, Photographs, Fig. 125 and Fig. 134, 'Phil. Trans.,' part 2, 1884.

[2] Experiment 27, Figs. 125–132.

[3] Experiment 28, Fig 134, l. c.

Professor Schäfer tells us that he too has observed hemiopia of temporary duration as the result of extensive lesions of the temporal lobe.[1]

It is evident that the hemiopia observed in these experiments does not indicate the position of the visual centres. For from these facts alone we should have as much reason for placing the visual centres in the temporal as in the occipital lobe.

There can be no doubt that the hemiopia in these cases was due to the lesion invading the occipito-temporal region, where the inferior occipital becomes continuous with the middle temporal convolution.

This point is situated on a level with, and opposite, the entrance into the hemisphere of the optic radiations from the corpora geniculata and primary optic ganglia.

It is to section of the optic radiations in this region that Professor Schäfer's results are to be ascribed. It is these which were destroyed completely in the case in which complete blindness occurred, and only partially injured in the case in which a large amount of vision was retained. Professor Schäfer cannot say, that the degree of vision retained by the latter animal depended on the cortex of the occipito-temporal region not being completely removed. For he relates a case in which, after complete recovery from any temporary effects caused by extensive removal of the occipital lobes, the destruction of the angular gyrus—*without any lesion of the occipito-temporal region in question*—caused complete and persistent hemiopia (p. 363).

Professor Schäfer has himself furnished me with so many facts showing that the visual centres are not confined to the occipital lobes proper, but embrace also the angular gyri, that it seems unnecessary for me to insist further on the subject.

Indeed, he appears himself to have some suspicion that "fibres connected with the cortex of the adjoining parts of the brain (and especially of the angular gyrus) may curve backwards into the occipital lobe, and thus become cut off along with that lobe" (p. 372).

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[1] "The nearer to the occipital lobe that any such lesion of the temporal lobe is carried, the greater probability is there of the occurrence of (temporary) hemiopia" (p. 372).

It is a pity that he did not examine his own facts a little more closely before committing himself as he has done. For he would have seen that the really effective lesions causing complete and permanent hemiopia—apart from conjoint destruction of the occipital lobe and angular gyrus—were those which severed the optic radiations in the position I have above indicated. And if Professor Schäfer will restrict his lesions to this region, without at all injuring the cortex of the angular gyrus or occipital lobe, he will obtain all the results which he attributes to removal of the occipital lobe itself.

While regretting for his own sake the attitude he has taken up on this question, I cannot feel otherwise than indebted to him for inducing me by his criticisms to re-examine, and, as the result, to re-affirm, with increased conviction, the views which I have previously enunciated.

POSTSCRIPT.

The Editor of 'BRAIN' has received the following note from Prof. Schäfer, with reference to an objection raised by Prof. Ferrier to the term "Report" used by him in the January number of this Journal:—

"A statement in my Paper in the last number of 'BRAIN,' regarding a report by members of a Committee of the Neurological Society having been understood to imply that a Collective Report had been published, I wish to state that the report here alluded to was an individual expression of opinion supplied to me by those members of the Committee (Drs. Bastian, Brunton and Waller) who took part in the investigation, and which it was agreed to substitute for a Collective Report."

ON A CASE OF LOCOMOTOR ATAXIA FOLLOWED BY GENERAL PARALYSIS OF THE INSANE.

BY F. ST. JOHN BULLEN.

Pathologist and Assistant Medical Officer to the West Riding Asylum.

A. C., admitted August 4th, 1884, into the West Riding Asylum. Æt. 24.

Statements in the certificate:

1. Has delusions of pecuniary and other possessions; is excitable and restless.

2. Has been altered in his mind since a "Paralytic Stroke" three months ago, becoming irritable and irascible, slovenly in his habits. Would go out, half-dressed, if not prevented. Memory defective.

Family History.—His mother in Wadsley Asylum, suffering from chronic melancholia. His father a most intemperate and passionate man.

History.—Patient's occupation had been that of clerk to an estate agent. He had led a fast and intemperate life, but became a total abstainer four years ago, and remained so until about one month before his admission. About three months previous to this, he was seized with a fit of the following description:—"Whilst sitting at his office desk and performing some calculations, he experienced a sensation as of a blow on the left forehead, and fell off his seat unconscious, and remaining so for about five hours. Subsequent to the fit, his right limbs, especially the leg, were partially paralysed. The act of manual prehension was, on both sides, inco-ordinate, his caligraphy extremely bad, articulation stammering and lisping. No aphasia existed, but a marked effort was required for him to utter words. All parts of the body were numbed."

These symptoms passed off in about three weeks, but he had suffered a great change in his moral character. He would go out in the street half-dressed, use abusive and threatening language to his wife, and, for several weeks prior to his admission, indulged in reckless debauchery. He complained after the fit of nearly continuous and severe cranial pain.

State on admission.—Bright, cheerful, conversable, and apparently of good intellectual powers. Explained the delusions mentioned in the certificate as "chaff," which he had uttered in response to some banter on the part of the workhouse porter. He denied any pecuniary or other possessions, but gave an exalted account of his mental abilities, saying he considered himself the equal of any living classical scholar, that he was Senior Classic at Cambridge in 1878, could speak seven languages and many dialects, and was a first-rate arithmetician. He manifested no failure of memory.

As regards physique, he was of medium height, rather poorly nourished; hair and irides dark brown; complexion sallow. Features clear cut, and expression intelligent. Both pupils dilated; the left nearly twice the size of the right, and the more active to light. Both reacted well to accommodation; sensory irritation produced no dilatation. His gait was somewhat staggering All the deep reflexes absent; superficial present. No want of tone in facial muscles, no defect of articulation. Respiratory, circulatory and genito-urinary systems were normal.

Progress of Case.—For the day after his admission he was quiet and orderly, but subsequently he became restless, interfering, abusive, unreasonable and excited; complaining after of much cranial pain. His statements as regards his powers as a linguist and classical scholar, etc., were found to be utterly untrue, and he admitted himself that his statements were "bounce." He was very untidy in his dress, constantly endeavouring to escape; had attacks of excitement, during which he would break windows, etc. After about three weeks of this behaviour, he became more reasonable and composed; no longer destructive; admitted all his previous boasting; made no more efforts to escape, and slept better. The left pupil continued the larger; in both, consensual, light, accommodatory and ocular movements were fairly performed. The tongue, protruded straight, was tremulous; speech shaky with an occasional trip in articulation. The gait markedly ataxic, unsteady, swaying to and fro; the feet lifted and set down jerkily and forcibly. Stood with eyes closed and feet approximated, he tottered and would have fallen if unsupported. No anæsthesia nor analgesia. He complained of difficulty in micturition at times; none in defæcation; also of frequent nocturnal seminal emissions. He stated that he had experienced diplopia both before and since admission, but more since.

After a lapse of three more weeks he became again excitable and emotional, declaring he was perfectly well; trying to escape through a window; was threatening. Complained still of the frontal cephalalgia. Soon after this, he spontaneously described severe lancinating pains darting through both knees, and mentioned anomalous sensations in his tongue and mouth, for which there were no objective signs to account.

The left pupil was now acting less well to accommodation than the right.

His importunate behaviour influenced his wife to take the responsibility of his home care, and he was discharged on November 13th, 1884.

He was readmitted on March 19th, 1886. Had been in the workhouse for some time, and in the certificate was mentioned as troublesome, quarrelsome, and complaining. On admission, the left pupil was still the larger one; both reacted to accommodation and consensually; the right, sluggishly to light; the left, not at all. The tongue was tremulous and the facial muscles also when he became at all emotional. Deep reflexes absent; the superficial blunted. Cutaneous sensibility apparently unimpaired from objective trials, but he complained of some numbness in the left foot. The calf muscles were atonic; gait markedly ataxic; progression performed on a wide base; heavily descending heels, and constant glancing at the ground and clutching at neighbouring objects for support. The lumbar curve was increased, a plumb-line from between the scapulæ falling one-and-a-half inches behind the buttocks. His handwriting was good, but a little jerky. Was unable to touch his nose with the index finger when his eyes were closed, or to bring the digits of the opposite hands approximately together.

Fairly quiet and free from troublesome propensities for about ten days after his admission, at the end of this time he again resumed his old querulous, complaining behaviour, making constant accusations against every one brought into contact with him. The lightning pains in his lower extremities were now frequent, and caused him disturbed nights; the seminal emissions were rarely present. About this time obscure dyspeptic symptoms arose. The pupillary movements were very incoordinate, especially on any attempts at convergence; ocular movements unaffected. He gave accounts of a momentary giving way of the right leg, accompanied with dimness of vision and confusion which occasionally befel him, and of his tongue being drawn to the right side of his mouth irresistibly at times, this accompanied with a sensation of its being too large for his mouth, feelings of chilliness and sickness and anomalous feelings about the face. Three weeks subsequently to the date of these last notes the lightning pains which extended down the thighs to the soles of the feet were accompanied with indubitable gastric crises. Occasional twitchings about his hands were noted, but these he stated had existed for nearly nine months. The succeeding month both lightning pains and gastric crises forsook him nearly entirely, but his complaining and accusing were constant; he developed delusions of suspicion and persecution on the part of the attendants, made entirely unprovoked attacks on helpless old patients, and at times had paroxysmal fits of passion, and indulged in foul and abusive language. Refused his food occasionally, but only for one or two successive meals at a time. Always intensely plausible and self-excusant.

Five months later the fulgurant pains returned, and now the upper extremities were involved; he described his legs as being shot out during the paroxysm, and complained of sharp, local pain over the area of distribution of Wrisberg's nerve at the elbow and of hyperæsthesia there. No superficial tenderness was present along the course of the nerve trunks, nor vaso-motor signs. He had occasional difficulty in emptying his bladder; the gastric crises had not returned at this time, but after two months' lapse they again set in; he could not sleep, even with the aid of chloral, and morphia which he had formerly taken to subdue the pain, he now rejected, persistently declaring that the medical officers were in league to poison him. He was excessively emotional, voice always very tremulous; his sight he described as not allowing him to read for more than five minutes at a time; the ataxia had so progressed as to render locomotion almost impossible.

The case now rapidly progressed to a conclusion. Rectal crises were superadded to the gastric, and these gave rise to fresh delusions on his part; he refused his food partially, and then, later, entirely. Fed once by the stomach-tube such distressing laryngeal symptoms were occasioned that its use was precluded for the future, and nutrient enemata were resorted to. He never spoke again, and remained in a pallid, cold, almost collapsed condition, with very feeble and irregular pulse. Required catheterisation often, and finally entirely. Ten days previous to his death, slight twitchings of the right side of the face set in, followed in twenty-four hours by severe general convulsions starting on the right side. These lasted about two minutes, and facial twitchings persisted for some time afterwards, but ceased under the influence of chloral nearly entirely. The next day he was able to swallow some liquid food; in the evening, however, the twitching again occurred, followed by a general convulsive seizure. During the next two days he had sixteen convulsive seizures; the day after, they were diminished by the use of chloral to four, the next, to one. He seemed in pain, moaning, and drawing up his legs in bed; constantly contorting his features. On the left side the mouth is spasmodically retracted, and there is considerable ptosis of the left upper lid; the pupil on this same side, as throughout, the larger. He now took a little liquid food by the mouth. Little change but for the worse now occurred; partly silent, partly moaning during the remainder of his life, he uttered from time to time a sharp piercing cry, which probably indicated a return of lightning pains; was always hostile. The spasmodic twitchings of the face continued; signs of vaso-motor paralysis on the right side were present; the temperature in the axilla 100·5, as compared with 100·2 on the left. Bed sores over the external ankle and sacrum rapidly formed, and death ensued.

AUTOPSY.[1]

Spinal Cord.—The membranes were slightly thickened in the lower portions of the cord, and more noticeably so in the cervical region. Here, too, the vessels were obviously enlarged and engorged with blood, and some, though not by any means marked, loss of transparency had taken place. The pia in the upper cervical segments was pretty firmly adherent.

The cord is small, firm and of irregular contour in the upper and middle dorsal portions. The lumbar segment is, in comparison to the cervical, much shrunken, indicating the further advanced condition in this situation of the lesion.

The posterior columns show disease throughout their entire length, of the nature of grey degeneration, quite plainly visible to the naked eye. Sections were taken at short intervals along the cord, both transverse and vertical, and were stained with aniline blue-black; Weigert's process was also used. The former method of staining gave as usual very perfect results, and rendered accurately every detail of the pathological change. The postero-median and postero-lateral columns are seen to be deeply sclerosed, the former in their whole vertical extent, the latter only partially, in their uppermost portions. The lesion also affects other portions of the cord to a less extent.

In the regions of irregular contour, viz., the dorsal, the anterior cornu of one side has suffered atrophic change and is reduced in width by at least, one-third. Its corresponding posterior cornu has likewise shrunken.

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[1] See plate and letterpress at the end of this article

An immediately noticeable feature is the greatly increased vascularity existing throughout the cord-length in the posterior columns and grey matter. Nor is it absent from the lateral tracts. A description of the entire vessel changes will be given later. In the lower portions of the lumbar segment, the posterior median fissure is open through its dorsal half; in the rest of the cord it is closed.

Lumbar region—Lower portion—Posterior columns.—The whole of these are deeply stained by the aniline dye, with the exception of the commissural zones, less affected, a tract of nearly healthy tissue lying between the posterior cornu and the diseased parts of the postero-external columns, and lastly a small oval area occupying the columns of Goll at the edge of the median fissure towards its peripheral extremity. This zone appeared quite healthy. The depth of staining in the diseased localities is due to connective tissue increase, both fascicular and corpuscular, to a quantity of amyloid bodies, to absorption of the dye by the myeline of the nerve fibres, and to a less extent to the increased number of hypertrophied vessels. Every degree of involvement of the nerve-tubule is seen from the healthy specimen to that in which the axis-cylinder is the sole remnant. In the peripheral parts of the columns, the normal structural characters are replaced by dense fascicular fibrous tissue, and here are crowded corpora amylacea. Immediately subjacent to this are scattered large medullated fibres, whose myeline has remained unstained. The great majority of the parenchymatous structure is extremely atrophied and distorted in shape by the shrinking of the interstitial tissue; often nothing remains but axis-cylinders darkly dotting the fasciculi of the latter. In the commissural ends of Goll's columns, the connective tissue is somewhat increased in the form of numerous islets from which branches radiate amidst mostly healthy fibres. The anterior portions of the internal radicular fibres could be traced with moderate ease; they showed an extreme varicosity in this part of their course. Much of the latter cannot be traced.

Sections of the posterior roots in their vertical axis show a great increase of the connective framework to have almost cancelled the nerve tubules. Where these are seen, they appear largely swollen in some instances, wasted in others; the axis not always visible.

Lateral columns.—There is a tract of sclerosis of triangular shape, one of whose sides is in apposition to the posterior cornu and whose base is at the periphery of the cord. From its basal portion is continued a marginal zone of degeneration which fades gradually and disappears at the anterior root zone. The place of the normal tissue is largely taken by close fibrous tissue throughout the above mentioned localities, and the septa, extending into the lateral column, are coarse and followed by numerous amyloid bodies, whilst young connective tissue cells are in abundance throughout the crossed pyramidal tract. There is not however here any obvious atrophy of the parenchyma.

Anterior Root Zones and *Cornua* appear free from disease.

Middle and Upper Lumbar Regions.—A progressive invasion by sclerosis of the posterior commissural zones is indicated by the increase of connective tissue and of capillary orifices. In the middle segment the small oval zone in the median line shows commencing involvement by the degeneration. A small artery surrounded by thickened pia passes on each side into it and delicate fasciculi of this connective tissue proceed between the nerve tubules. Appearing between the latter, and displacing them, are branching cells whose processes spread and anastomose in every direction. At this stage no primary affection of the nerve fibres was in any way apparent.

In the upper lumbar segment, it is seen that this zone is now completely involved in the prevailing lesion, but extending backwards from it along the edge of the fissure is a narrow bordering of fairly healthy fibres. The commissural ends of Goll's columns manifest a marked increase of both connective tissue and capillaries, the latter usually forming the centre of an islet of the former. A large number of nerve-fibres have undergone change and the neuroglia is often finely dotted with the axis-cylinders. The corresponding part of Burdach's column is less involved, but there are a number of diseased fibres scattered about the field, independently at this level, of increase of neuroglia.

The marginal degeneration of the lateral columns gradually lessens from the lower segment of the lumbar cord upwards and in the sections cut from the lowest dorsal region is not visible. There is a progressive increase of interstitial tissue in the crossed pyramidal tract.

Lower Dorsal Region.—Posterior columns. The degeneration is still very intense, and as in the lumbar region involves in the greatest degree the periphery of the cord. The vascular changes are more evident than as yet seen, and capillary orifices are in great number, and the postero-internal and external arteries are enlarged and tortuous. The median commissure is freely channelled by large thick walled vessels.

The amount of neuroglia in the posterior commissural zones is somewhat decreased, compared to that in the last segment of the cord described, but is thrown into greater prominence by the larger number of healthy fibres present.

Anterior columns and grey matter. These showed changes of the same nature as, but to a less extent than, those presently to be described.

Upper Dorsal Region.—Posterior columns. As compared with the rest of the zones at this level and its corresponding area in the lower dorsal segment, the tract including the periphery of the columns, the posterior nerve roots and the part of cord just internal to them, is less degenerate, and the lesion is tending to centre itself in the columns of Goll. In the commissural zones the number of healthy fibres is again reduced. The connective tissue is comparatively and relatively in excess over that in the upper lumbar region, and forms the predominating element in the field. The result of its contraction on the nerve-tubules is seen in two forms; atrophy as elsewhere, and a large swelling of the fibre which becomes indistinctly defined, of a dull stained appearance, and its axis swollen, wanting clearness, often hardly differentiable. These bulgings of the tubule are usually surrounded by an especially dense zone of connective. These appearances of degeneration extend into Burdach's columns, but are less in degree and area. Here and there are isolated tubules, or small groups of tubules, affected seemingly independently of neuroglia increase, but it is probable that these have been strangulated at a lower level.

Lateral columns. These presented in the region of the crossed tract the same indications of lesion as before detailed.

Anterior columns and grey matter.—The anterior and posterior cornua on the left side have undergone marked alterations, and the corresponding hemisphere is changed in contour, much flattened, and its transverse diameter considerably diminished. The left anterior horn is somewhat longer than its fellow, lessened in width by fully one-third, its summit club-shaped instead of square. The extensions from its outer side are almost absent; from the inner, very sparse. Hardly one healthy ganglion cell is visible; such as are seen are in all stages of atrophy, shrivelled, apolar and situated in vacuolar spaces, not seldom the latter are the sole indications of their former presence. The border of the posterior horn and intermediate tract is with difficulty defined, the grey matter merging into the white. The cinereal substance as a whole is friable-looking and broken up; so loosely combined is its texture that under a low-power it has a peculiar patchy-white unstained appearance. Both cornua are freely sieved by numerous ascending capillary vessels, whilst the substance of the anterior is much channelled by its enlarged arteries. The cells of the posterior cornu are also constantly shrunken and apolar, no trace of branching connections can be seen. In consequence of the atrophy of the outer part of the anterior, and the indistinct definition of the corresponding portion of the posterior horn, the two appear to lie nearly in a straight line. Clarke's vesicular column on this side had a friable and degenerated appearance, being involved in the lesion of the grey matter, and its cells are shrivelled, often extensively pigmented and many wanting. In a number of sections taken, the average drawn was left to right as twenty-nine to forty. The invasion of the groups occurring from the outer side; it is here that the remains of destroyed cells are mostly seen.

The posterior horn on the right side is far from the normal, its appearance simulated less markedly those on the left. The right anterior horn is deeply stained, but the contour is not obviously incorrect. The cell groups in this region are normally most indistinct, and the number of cells sparse, but comparison with a healthy specimen favoured both a decrease and deterioration of them here.

The column of Turck on the left side was much smaller than that on the right, its comparative diminution being in about the same ratio as that of the corresponding cornu. There however, beyond this, was no obvious disease of its elements, parenchymatous or interstitial. Such condition was present from the lowest dorsal segment upwards, and in the decussation region is still to be traced.

Cervical Cord.—Four segments of this were examined.

Posterior columns. In the first and lowest the clearing up of the degeneration in the periphero-external part of Burdach's column is progressing. Goll's columns are densely sclerosed still, and at their commissural ends the increase of connective tissue appears greater than any yet seen, and extends right up to the commissure. This portion of both columns is sieved by the numerous capillary orifices.

The lesion in the two higher segments presented characters generally similar to each other, and those of one, the upper, are alone detailed.

The postero-internal half of Burdach's columns is nearly normal, a few atrophied marginal fibres, an increased vascularity and numerous corpora amylacea only constituting morbid signs. The internal half of its periphery shows many fibres, enormously swollen, whose axes are feebly stained, greatly hypertrophied, often lying at the extreme side of the tubule, even not visible. These are scattered amongst nerve fibres some of which are extremely atrophied, others compressed, mis-shapen, stained, and ill-defined. In the commissural parts of these zones a narrow strip of comparatively unaffected tissue separates the grey matter from the advanced degenerative changes. There is, however, even here, some excess of neuroglia, and a few large vessels are seen bounded by healthy fibres.

The degeneration in Goll's column extends up to the commissure; its characters differ somewhat in parts, as now described.

In the posterior third, roughly speaking, the changes presented are those of universally wasted and degenerated nerve fibres, irregular in contour from compression by the fine fasciculi of connective tissue which are closely specked with the axes of the degenerate smaller tubules. In about the middle third there are many medium and small-sized fibres of fairly healthy appearance, whilst areas of intense lesional alteration of larger and smaller extent exist.

The degeneration in the commissural portion is very advanced, and the changes shewn are of a different kind. A darkly-stained ground of utterly degenerate fibres and excess of neuroglia is mottled with bluish-white patches or streaks formed by partially diseased tubules the myeline of which is still preserved though stained a little. The nature of these streaks is only discoverable on careful focussing, the refraction being altered. Scattered about are large pale, oval, or round discs; probably fibres undergoing colloid change; the axis can but seldom be brought into view. Lastly, there are a few tubules of approximately healthy appearance.

Vessels in number traversed the field both vertically and horizontally; those at the marginal parts of the diseased areæ are as, before, the centre of an islet of connective tissue.

The sclerotic shrinking of the posterior columns has torn open in many places the septum closing the median fissure, and this is also the case with the division between the postero-median and external columns. In these situations the arteries are much hypertrophied and their lymphatic sheaths crowded with cell contents, and the processes of pia into the cord are infiltrated with leucocytes.

Upper Cervical region.—Lateral columns. The crossed pyramidal tract is stained in excess of the usual deeper colouring of this region, owing to an increase in the amount of connective tissue and blood vessels. There is a tract of somewhat densely fibrillated tissue lying in front of the posterior cornu, which, passing outwards, extends along the periphery of the cord, narrowing gradually; opposite the level of the tip of anterior cornu it disappears. From it coarse septa extend into the column, numerous amyloid bodies accompanying them. Large deeply-stained branching cells extend their processes between the cross-sections of the tubules in the pyramidal tract, and contiguous to them are fibres showing commencing change. The most noticeable feature in the field is presented by the vessels. They are scattered in considerable number throughout the whole lateral column, excepting its marginal portions, but are especially congregated in front of the posterior horn, and laterally to the anterior horn. Here the substance of the cord is very freely channelled. The appearances in the two lateral columns are not quite symmetric, the left not showing as much connective tissue increase, though no appreciable difference in the number of vessels. Flechsig's limiting zone is quite intact.

Anterior cornua and columns.—In the lower two segments of the cervical cord, pronounced changes are seen in the grey matter. The anterior horn of the left side is narrower than the right, and instead of a bold convex outer contour, is concave from the falling in consequent on the disappearance of the antero-lateral group. All the cell groupings in these segments were very indistinctly marked, and the cells for the most part shrunken, black, and with processes stunted, broken off or absent. The internal on both sides is less degenerated than the others; the antero-lateral is more affected, whilst the postero-lateral group is almost completely wanting. The cells in the central area are hardly distinguishable. These atrophic changes were nearly symmetric, but the right horn was a little the less involved. Many coarse vessels traversed the cornua; the cell groups are often, quite indifferently, broken up by some large branch, and it is observable that the higher up in the cervical cord sections are taken, the more does the vascular increase affect the grey matter.

Moreover another feature to be noticed is, that although there is throughout the greater vertical entirety of the lateral columns a marked excess in the vessel numbers, together with hypertrophy of their coats, yet nowhere is there any sign of accumulation in the perivascular sheath of lymph-cells, or such changes in general as are met with in connection with the vessels in the posterior columns. The central canal and the grey substance immediately surrounding it presents a heaping-up of dark-stained endothelial cells, the canal being in much of its extent obliterated by them—this appearance applying to it in the entire length of the cord.

In longitudinally-cut sections of the posterior columns the medullated nerve-tubules are, more markedly at the periphery of the cord, of an extremely varicose appearance. This not seldom seems due to constriction by embracing fibrils of connective tissue, but more often independently of such a cause. The axis-cylinder is, at times, swollen and fusiform for a small part of its length, very tortuous, and like the nerve-tubule presents marked varicosities and constrictions.

Addendum.—It was not found possible, at the date when the description of the lumbar cord was written, to procure sections from the extreme lower end in that region, owing to the friability of the tissue. By the use of a modification in the imbedding process, later on, satisfactory specimens were obtained. The sclerosis here is seen to be less diffuse, and to admit of localisation better. The internal radicular fibres appear to constitute especial lines of intensity; elsewhere it is perceptibly gathered around the vessels in their course, and these latter form a far more important feature in the field than they should in a healthy cord, in this region. The bordering of healthy fibres along the posterior median fissure extends further towards the commissure than in the sections of the lumbar cord described at the commencement of this paper.

Vascular system.—Its condition has been briefly alluded to as the various cord-segments have been described, but some additional notice may be given to this. It may be said, broadly, that scarcely a healthy vessel can be seen throughout either cord or medulla; this statement however involving the acceptance of hypertrophy and dilatation as evidences of departure from the normal. Such conditions as the latter are present, or perhaps frequent, in portions of the cord where no obvious disease of its structure exists, and, beyond having a certain bearing on the question of a general vascular lesion, do not call here for more remark. It is in the posterior columns and the grey matter that a change of interest has occurred.

In the commissural zones and grey matter this increase takes the form of very numerous pin-hole apertures of circular, defined, or punched-out appearance, the transverse sections of capillaries. These are in greatest number where the sclerosis is younger, and seemed in many parts the evident precursors of the degenerative change. In the remaining area of the posterior columns the vessel orifices are less numerous but larger, and the majority of the arteries are met with running inwards in the plane of the section. The most prominent of these in the lower segments of the cord are the postero-internal and external arteries, which are greatly hypertrophied and enlarged, cut in more or less of their extent; that part of the former which lies along the posterior cornu is even more enlarged than any other, and is so in the lowest sections taken. The smaller vessels in this region have their walls collapsed and lumen obliterated by the shrinking of the sclerous tissue; in the higher parts of the cord the increase in number of the arteries is more readily seen. The median fissure is occupied by a large branch and an also large offshoot from this runs into Goll's column on each side.

In the grey matter of the posterior horns, capillary orifices are extremely numerous; that of the anterior horns is traversed frequently by large channels; seldom, nearly bisected by one. The commissures show groups of vessel mouths, and are almost interrupted by channelling in many sections. Occurring also with tolerable frequency are large circular excavations, probably the result of vessel changes.

In sections cut in the longitudinal axis of the cord, the posterior columns appear almost riddled with apertures, vacant or containing the remains of vessels. These openings are for the most part much in excess of the diameters of the latter, but their contents in these sections are mostly absent; around the margins, of dense sclerosed tissue, are numerous connective tissue nuclei and amyloid bodies. In the parts of the section corresponding to the commissural zones, the arteries seen in length are occasionally of a remarkably varicose outline, suggesting the affection described by Obersteiner as occurring in paralytics.[1]

Synopsising the vascular changes found throughout the medullo-spinal axis, the arteries are remarkably often tortuous; their coats much hypertrophied, the lumen sometimes dilated; sometimes invaded, even to obliteration, by conjoined thickening of the walls and contraction of the surrounding tissue. This thickening of the tunics is not uniform, being generally increased at the points of curvature, probably both from approximation of naturally more distant points, and from actual increase in the amount of tissue. The cut edge of the vessel has a homogeneous, ground-glass-like appearance as a rule.

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[1] 'Rosenkranzform des Muscularisrohres.'

In parts the adventitial coat has been drawn out, and is seen to be of a finely fibrous texture and thickly sprinkled with branching nucleated cells on its inner surface. The endothelium is pretty generally swollen and indistinct, and its cells detached often in mass into the lumen of the vessel.

Along the course of some of the smaller arterioles are seen young branching connective tissue corpuscles. These not only lay outside, but inside the perivascular sheath; in the latter situation in company with embryonic cells, their processes passing outwards between the nerve fibres. Throughout the cut edge of the vessel coat, cells with or without off-shoots are seen lying, occasionally protruding in part from the outer surface of the adventitia, or passing through the endothelial lining. In the last, are imbedded leucocytes at intervals.

The connective increase would suggest an origin in a proliferation of the cells of the adventitia, and in migration of lymph-cells through the vessel coats.

In the dorsal portions of the cord above them, these changes in the perivascular sheath are replaced by others of a marked nature. It is distended by large, pale, granular, unstained bodies, round or ovoid shaped; the nucleus in which is of almost equal size to that of the cell itself. With these in the space are embryonic cells, pigment and granular masses.

In the cervical segments, the accumulation is remarkable, and not limited to the perivascular space, but existing between the arterial tunics and splitting them up for a considerable distance, or else heaped up in limited areæ causing a local occlusion of the lumen. These bodies are apparently, in some instances, diffused into the surrounding tissue.

Throughout the medulla, pons, and crus, are met with, here and there, vessels whose outer border has a strikingly beaded appearance and double contour. Under a high power, such characters are due to a single row of fairly regularly-sized small round bodies in the perivascular lymph-space.

The other vessels in these regions show frequently cells of less regular shape in the same situation, and less in number, or merely thickening of their coats, and cell proliferation.

Medulla.—The upward progress of the degeneration along the cuneate and slender columns is plainly visible, and the cells of their nuclei evidently atrophied and otherwise diseased.

The fibres constituting the zonular layer show throughout their extent a varicose outline, such as is seen in those of the posterior columns. Thickly scattered along their course are amyloid bodies and connective tissue nuclei, the distribution of these being continuous from the posterior columns into the nuclei of the pyramids and these latter indicating changes in excess of the allowable variations in healthy tissues. From these nuclear formations connective tissue nuclei follow the course of the fasciculi passing into the median raphe; there are they lost in the deep-staining of the latter, and no disease is traceable along its extent, but those fibres from its posterior part which penetrate the hypoglossal nuclei manifest tumefaction and varicosity to an extreme degree.

Those inner bands of the arcuate system, which, skirting the central grey column are to be traced into the pyramids, appear altered from the normal. They are narrower and their medullary sheath wasted, the fibrillæ having a thin, thready look; their outline is blurred and indistinct, by reason of the absorption to no small extent of the aniline dye.

In the lower regions of decussation, the sclerosis extends from the posterior columns around the caput cornu, investing it; higher up its position is occupied by the ascending roots of the fifth, and these latter, whose sectional area always presents a dark appearance from the scanty white sheath possessed by the tubules, are traversed by a considerable excess of fibrous tissue. The same increase of neuroglia and a complete, but not uniform, obscuration of the nerve-fibres is seen in the funiculus solitarius on both sides, and the fibres of those nerves which curve round this, their column of origin, are at this point remarkably varicose and swollen.

The central grey substance, in the region of the inferior pyramidal crossing is much channelled by vessels. Here also a large transversely running fissure is placed just posterior to the central canal, completely bisecting the grey matter, and probably resulting from the friability of the latter produced by degeneration. Not seldom large cyst-like cavities appear; their nature in some is indicated by the adherence to their walls of portions of thickened vessel tunics.

The group of cells occupying that portion of the central grey substance placed postero-laterally to the canal is on one side degenerated; very few of the cells remain; they that do are shrunken or inflated, mostly apolar.

The ganglion cells of the anterior cornua and those scattered throughout the reticular formation, seem healthy, also those of the restiform body, hypoglossal, abducent, and facial nuclei.

The vagal nucleus of one side is degenerated; the tissue in which it is situated has the same whitish, friable look as that of the cornu in the dorsal region. Through several sections its cells are seen to be in different stages of decay, and compared with those of the opposite nucleus to be greatly diminished in number, the number also of those always pigmented appears decidedly increased.

In some successive sections is a large tortuous vessel, occupying the centre of the cell-group and breaking it up.

The affection of the corresponding vagal nerve is by no means so marked as that of its nucleus, but by the observation of a number of sections morbid changes in it became certain.

The area of its fasciculi is diminished, and the latter are often deeply stained; those fibres which join the main trunk from the substantia gelatinosa are especially changed, and their medulla appears almost completely wasted. The moniliform character of the diseased nerve tubules is very noticeable. At the border of the medulla, where the trunk crosses the zonular layer, its fasciculi are invaded by the connective tissue corpuscles and amyloid bodies which stud the latter as far inwards as the gelatinous substance.

In sections taken through the upper planes of the medulla the acoustic nerve and some part of the dispersed nuclear origin is diseased. Unfortunately one side of the section had lost the nerve root; it was impossible to identify which. The sclerosis in the acoustic trunk is extreme; its area is composed of thick wavy bundles of connective tissue, the inter-spaces of which are occupied by almost unrecognisable remains of nerve-fibres imbedded in the coarsest meshwork of neuroglia; in the septa are running numerous dilated vessels. Those of its root-fibres passing between the gelatinous substance and the inner peduncular body are deeply sclerosed, those peripherally situated are less affected and signs of disease in them fade fast towards the nucleus. In several sections a long and coarse artery traverses the nerve-root for some distance. A strip of pia, adherent in all specimens, between the nerve and the body of the medulla, is seen to be considerably thickened, its vessels large and engorged with blood, and a number of leucocytes to be scattered in its meshes. The cell group of the posterior median acoustic nucleus lying to the inner side of the internal peduncular tract, it was easy to assure oneself in ten sections was degenerated, one being invariably correct in identifying the diseased area as to its side without the aid of the corresponding nerve. The change has mostly affected the large multipolar cells, which are shrunken and their processes stunted or absent.

The uppermost portion of the internal auditory nucleus, a small clump of cells lying beneath the grey floor, appears on the one side free from lesion, on the other it was entirely absent.

There is an increase of connective tissue in the pyramidal columns of the medulla in its lower planes, but it is gradually lost in the upper, and no lesions, except those described, are apparent.

Quadrigeminal region.—The tissue in this region is rendered so friable by disease that satisfactory sections are difficult to obtain. The peripheral part of the specimen on the left side has been injured a little during the course of preparation, no important structures are however damaged, and the respective sides of the mounted sections are made by it capable of certain identification.

The vascular increase here as elsewhere is very marked. In the mid-line behind the aqueduct is a large thick-walled vessel, which may be said to represent the artery of the posterior median fissure. In the sections in which this vessel is not present, its position is occupied by a deep-stained band of fibrous appearance, on and around which are thickly-dotted amyloid bodies. These latter are very numerous throughout all the layers posterior to the aqueduct.

Although the entire medullary matter is over-vascular, yet this latter condition is more especially marked in its left half. Here a complete riddling of the tissues by thickened vessels has occurred.

In the anterior half of the left side of the chain of fibres constituting the descending roots of the fifth nerve, the fibres are much swollen, but clearly defined and unstained; the axis cylinder, however, cannot be detected in one of them.

The oculo-motor nuclei at the level of the nerve roots show considerable changes. The cells which are scattered amongst the fibres of the raphe are very few and deeply pigmented; the smaller elements which lay most posteriorly are still more obviously diseased, whilst the median and largest group is asymmetrically affected. On the right side its cells are thinned in number and atrophied, on the left the group is split up by a large coarse vessel which runs the whole anterior-posterior extent, and on each side of which the nervous structure is plainly diseased, but far more on the inner side, where the nerve-cells are almost absent.

The disease in this nucleus, although tolerably uniformly distributed, is intensified around the vessels, and the latter suggest themselves as an evident origin of the lesion. Both posterior divisions and the left median of it are especially invaded.

The dispersed cell formation, existing in the grey matter anterior to the aqueduct and below the level of the roots of the third, is prominently degenerated. Almost all the cells are extremely pigmented in a large number represented only in granular masses.

The fibres of the third nerve do not appear consistently diseased, but in some sections are fairly healthy-looking; in others the bundles are very meagre and threadlike.

Owing to the condition of the grey matter of the quadrigeminal bodies, the characters of its cells are rendered somewhat uncertain. The larger form, whose processes lying radially are understood to assist in the completion of the reflex arc between optic and third nerves, where distinctly seen do not appear diseased.

The Optic Nerve.—The sheath of this nerve is considerably thickened and the septa of neuroglia very much so. The latter appear as coarse, wavy, connective tissue, freely permeated by vessels, infiltrated in the vicinity of the latter by leucocytes and branching into the nerve bundles in all directions. These last are also invaded by arteries independently of the septa, and are plentifully dotted with an increased number of somewhat enlarged connective nuclei.

The external layer of some of these arteries has undergone so much thickening and its laminæ have become so wavy and differentiated as to resemble very closely the processes of connective tissue. The perivascular sheath almost invariably allows its contents to be recognised, in the form of swollen, largely nucleated, endothelial cells and lymph corpuscles. The vessels themselves are mostly engorged with blood or filled with disintegrated endothelium. In some cases their walls show the change noted elsewhere, in the description of the vascular system, viz. the beaded contour. This, as before said, depends on a line of round lymph-cells in the perivascular canal; it is quite distinguishable from any appearance produced by foldings or localised thickenings of the vessel tunics, in that certain segments of the canal shew a double or triple line of these bodies, and others a rupture of the sheath and a discharge of them into the space around. Arteries with these localised thickenings are elsewhere to be seen. In spite of these hypertrophic changes, vascular and interstitial, the bulk of the nerve is reduced by reason of the atrophic changes in the nerve tubules. These are closely compressed, cannot be distinctly defined, and show a coarsely granular appearance. These signs of degeneration are present in all the bundles in the central portions of the nerve, towards the periphery are less marked, and at the latter part the nerve tubules are generally swollen, varicose and ill-defined.

The Sciatic and Anterior Crural Nerves—were examined on both sides and all showed changes of a similar kind. They have a spongier and less resistant feel than that of healthy specimens, and this is seen under the microscope to be due to the large amount of adipose tissue existing throughout the nerve, widely separating the groups of nerve bundles. The epineurium is invaded almost universally by this fat increase; in parts, its proper structure is practically replaced by fat-cells, everywhere the latter are present in it to some extent. When connective tissue-cells can be made out, they are seen to be distended with oil globules. The endoneurium has undergone marked hyperplasic change; it appears in the form of coarse, wavy, deep-stained bands of fibrous tissue spreading across the nerve bundles and obscuring many. The larger tubules, in all the nerve-bundles, seemed to have escaped change, those suffering being the smallest. Nor are the alterations at all uniform throughout the section of the nerve. Many bundles appear but little affected, whilst, in others, especially at the periphery, a very marked wasting of most of the fibres has taken place, the area of the funicle being composed of deeply-stained fibrous tissues and the remains of atrophied tubules, dotted towards the marginal portions mostly, with clearly defined uncoloured fibres. Rather seldom are seen very small bundles of atrophied tubules situated in the midst of adipose tissue, presumably the remnants of larger and healthy ones.

The vascular increase through the nerve is large. The arteries are possessed of remarkably thickened coats in which no detail of structure can as a rule be made out; they are both very numerous and much enlarged; are either engorged with blood corpuscles or blocked by a mass of welded endothelial cells.

Brain.—The skull-cap is symmetrical, rather over the average thickness; very dense, hard and heavy. The dura mater is very loose and baggy over the frontal regions; somewhat congested and pretty firmly adherent to the general cranial surface; its appearance otherwise normal. It is free from any cohesion to the inner meninges, except along the vertex at the sites of Pacchionian bodies. The superior longitudinal sinus contained throughout pale, firm, clot, the lateral dark soft clot. The subdural space contained some excess of thin, reddish, slightly turbid serous fluid.

The brain on removal appears of fair size, but narrowed in the frontal regions; its consistence is much, generally reduced, and placed upon the table; the hemispheres separate widely, uncovering much of the cerebellum.

The arachnoid over the fissures and sulci is buoyed up by fluid between it and the pia; over the summits of the gyri, closely applied. It is thickened and a milky opalescence is observable more or less uniformly, but especially along the course of the vessels. The pia was of increased consistence and hyperæmic. The membranes as a whole are increased in thickness and toughness, and strip in sheets from the brain. Their removal, performed with great care, is accomplished in spite of the diminished consistence of the cortex without its laceration, except at certain sites of adhesion to be detailed. Here the surface is left eroded in narrow patches, presenting a characteristic worm-eaten look; for the most part in the midline of the summits of the gyri, else placed a little laterally; always however at their free surface. The cortex in the sulci is perfectly smooth. The sites of erosion are the following: about the middle of the superior and middle frontal, the inferior extremities of third and ascending frontal, and the median extremity of the latter, all on the left side. On the right, the erosion is much slighter and more superficial, and appeared in two or three scattered patches over the first and second frontal gyri.

On removal of the membranes and consequently of their supporting influence, the softened brain loses all its contour and becomes flabby and flattened out, and its manipulation is far from easy. The convolutions are considerably atrophied, especially the frontal, and the intervening sulci gape widely. The gyri are of good average complexity and normal arrangement.

The basal vessels present no abnormality of structure or disposition. The grey cortex is lessened in depth, pale, and indifferently striate. The white substance much softened, œdematous and tenacious; its puncta very few. The basal ganglia are pale, look ill-nourished, and their vessels very coarse and gaping; neither on the right nor left side is any focal lesion present, nor any trace of such. The ventricles are somewhat distended with reddish fluid; their ependyma appears smooth. The optic nerves and tracts, crura, pons, and medulla, are reserved with portions of the cortex.

Total weight of brain, 1310 grammes; right hemisphere, 550 grms.; left do., 445; cerebellum, 160 grms.; pons and medulla, 30; cerebrospinal fluid, ℥ iv.

Cortex Cerebri: Frontal gyri.—The sections prepared by the fresh method appear to have been made at the site of decortication only, since the outermost layer of the cortex is not preserved. The second and third laminæ show changes, more marked in the latter by reason of the larger size and wider separation of its nerve-cells, of the following nature. There is a large increase in the number of the free nuclei, (this existing through all the strata); obscuration of the ganglionic cells by round bodies (lymphoid?) which lay both outside and over them; a substitution of two, three or more elements similar in appearance, for the angular nucleus in the pyramidal cells, and lastly a distortion of the shape of many corpuscles owing to atrophy and apolarity. These various conditions contributed an appearance of crowding, irregularity and indefinity to the ganglionic formation.

The apolarity of so many cells is most probably not merely an effect of uneven section, but an actual loss of processes, since these lie amongst other corpuscles whose poles pursue a long course in the field. It is also noticeable that those cells preserving their polarity, retain almost invariably an intact angular nucleus, whilst those in which the latter is replaced by several rounded elements are they whose processes are most often lacking. Moreover it is observable that the former class are very seldom surrounded by the nuclear bodies, so that the latter would seem to be associated with the condition productive of atrophic changes in the cell and segmentation of its nucleus. These rounded elements are often seen in the pyramidal cell formation of senile brains, but under ordinary circumstances not in such number as are here present in this patient; and this, in conjunction with his age, indicates their import.

In no stratum of the cortex is this condition of obscuration of the cell by nuclear elements so marked as in the claustral. The large number of the corpuscles are entirely concealed by grape-like clusters of them, from which, sometimes, a short process projects like a stalk. Of this formation the cells less affected seem to be those whose axis is vertical.

The increase in the number of vessels in this layer is perceptible; their walls are closely studded with nuclei, and often they appear to be represented by lines of aggregate elements of the same nature. Here and there, bud-like excrescences of these project from them, and not infrequently is there evidence of an active migration of leucocytes.

In the hardened sections, care was taken to cut through the layers of the cortex in the vicinity of the eroded portion as through those of the latter. The first layer preserved in these specimens shows a notable increase of the neuroglia both in the form of fibrils which give a fine mesh-work appearance to the ground-structure, and of young spider cells. The latter are numerous, and are present in no other stratum of the cortex save the first.

The vessel coats are thickened, tortuous, and wavy; the lumen of the vessel is often choked with epithelial débris. The perivascular space contains rounded, granular, or pigmented bodies, or nucleated endothelial cells. These products are occasionally seen in less amount in the subadventitial space itself. In a few instances the arterial tunics are seen lying shrivelled up in a space occupied by a compressing mass of welded endothelial cells.

COMMENTARY.

The symptoms of Tabes were so well marked in this patient that any doubt as to the nature of the case is not possible. Those of the mental disease are however not so defined, and will need further discussion.

Before considering this subject, there are some points to be touched on in the pathology of the disease worthy of mention.

With reference, firstly, to the upward continuation through the medulla of the morbid changes in the cord. At the uppermost part of the latter, the sclerosis was most pronounced in Goll's column and the funiculus gracilis, in accordance with this, has suffered mostly. The disease in the root zone is represented, though to a less extent, in the respiratory fascicle; and the ascending root of the 5th was degenerated. The unilateral involvement of Clarke's vesicular column in the mid and upper dorsal region of the vagal nucleus and the cell-group in the grey matter at the inferior planes of the medulla, which probably represent it, help to draw inference not only as to mutual relationship, but as regards function, the gastric disturbances being considered.

The predominance of the changes in the grey matter and cell-groups from the dorsal region upwards on the left side is interesting.

The appearance of the cord in some respects indicated perhaps a congenital defect of organisation; were this so, it might account for the more ready succumbing of the tissues of that side to disease.

The descending fibres of the fifth nerve were unilaterally (on the left) and partially affected; their possible relationship to early diplopia has been pointed out by Dr. Ross. It will be remembered that in the present case the latter affection was said to have been present for some time prior to admission, and that the fibres are diseased on the side of the larger pupil.

The bilateral mydriasis present when the patient first came under observation, may be perhaps connected with the sclerosis in the cervical region, since sensory stimuli failed to arouse further dilatation.

Accommodatory movements were not moreover affected at this time.

Later on, in the progress of the case, the ocular symptoms were, entire loss of light reflex in the left eye, partial in the right, consensual and accommodatory movements being preserved. The consensual reflex in both eyes demands the intactness of one optic tract, of the commissural fibres between the oculo-motor nuclei, and of those portions of the latter, at least, which subserve the function of light reflex. Since vision was preserved in both eyes, at any rate not seriously damaged, the optic tract must be allowed to be open for impressions.

Again, since consensual movements were present in both eyes, the light reflex portion of the oculo-motor nuclei cannot have been seriously involved. The remaining tract is that which completes the reflex arc, a function whose morphological correlatives are supposed to be Meynert's fibres in the quadrigeminal region.

Such parts of these as could be seen in the latter did not appear diseased. Only for a limited portion of their course however were they observable. The superficial and partly the deeper medullary layers were extensively broken up by vessels on the left side, the right was also, but to a less extent, the sufferer by a vascular increase. The condition of the consensual reflex is not unfortunately noted after this just quoted stage in the ocular troubles, the loss of accommodative contraction being alone commented on. But from the pretty general affection of the oculo-motor nuclei it could hardly have been preserved. An interesting and valuable case is reported by Dr. Ross, in "BRAIN" (April 1886), in which a definite lesion of these fibres which travel betwixt the anterior tubercles and oculo-motor nucleus was discovered. Though owing to the diseased condition of the tissue in this region, my specimens were not sufficiently distinct to shew this, or such a lesion as would have produced the same effects, yet the morbid signs would have rendered the preservation of the function of the fibres quite as remarkable as its destruction.

In any case the maintenance of consensual reflex still further localised the disease.

The irregularity of the pupils suggests a disturbance of sympathetic innervation or of the influence of the meningo-encephalic adhesion existing over the middle portion of the second frontal gyrus. In connection with this we may note the fact, that the convulsive twitchings mainly affected the right side, or that opposite to the site of adhesion and the dilated pupil; and that in the intervals of actual convulsive seizures, a constant retraction of the mouth occurred with occasional sharp jerking of the head to the right side. A progressive invasion of the ocular-motor nuclei is indicated by the loss of contraction with accommodation, and the inco-ordinate movements on convergence efforts. After the convulsions, were ptosis and external strabismus on the left side. The commencing loss of accommodative contraction is noted at the same time as the advent of the lightning pains; that of light reaction with the blunting of cutaneous reflex. Regarding the lesion of the acoustic nerve and nucleus there is but little to say. It is probable that the condition of such parts of the receptive mechanism as were involved, would likely influence, in a structural or functional modification, the receptive centre. The patient never complained of deficient or anomalous perceptions of sound, nor was the presence of aural hallucinations ever suspected. It is however just possible that such may occasionally have existed in some form, from the impulsive nature of some of his acts. He would suddenly abuse his fellow patients and the attendants without cause, and often strike at the former, always, though, choosing the aged or helpless patients. His frequently and plausibly urged excuse was that they had insulted him or called him names.

It is remarkable, when the extensive lesion of the lumbar cord is considered, that there was no coarse impairment of sensibility, which there was not, since the patient was perfectly able to recognise the nature of the substance on which he was standing, and to localise pin-pricks, etc.

Allusion may briefly be made to the greater wasting and decortication of the left hemisphere and the limitation of the latter to the frontal regions on both sides, in connection with the type of the symptoms. Also to the occurrence of vaso-motor paretic signs and elevation of temperature on the side of the convulsions.

There is so much difference of opinion regarding the true nature of Locomotor Ataxy, systematic or neural, diffuse or perineural, that a short discursion as to the validity of certain observations, brought forward in evidence of a definite origin may not need apology.

A fact of great importance in the estimation of tabes as a systematic disease, is adduced by certain authors (Strumpell, Westphal), viz. the peculiar localisation of the degeneration in certain areas of the posterior columns.

That such localisation may be made in the earliest stages of the disease is probable, but that they are proof of a system fibre affection, I do not see sufficient evidence to accept at once.

A difficulty in the investigation of the lesion in the initial periods of the disease, lies in the rareness with which a pathological examination can be made at that date. Without wishing to assume any identity or the reverse between the cord lesions in tabes, and those in cases of general paralysis with ataxic symptoms, I have nevertheless taken advantage of the opportunity such cases offer for the early observation of the degeneration of what nature it may be, in order (as one of the many objects of such a research) to discern any especial localisation to some defined area of the lesion, on its commencement. I have examined sections from the various regions of the cord in eight cases, and in the majority, the degeneration has been in a very early stage, and allowed of its characteristics being easily defined. I quote, very briefly, only such results as directly bear upon the question of primary localisation now treated of. Two prominent pathological facts in all the cases at once appear. They are a primary increase in the perineural tissue and a multiplication and enlargement of the vessels seen. Of the precedence both have to any nerve fibre affection the histological characters leave no doubt. I do not intend to enter at any length at all in this paper into the mutual relationships existent between vascular and neurogliar increase, but it may be said briefly that the vessels served as lines or centres for the development of the tissue around them. This was obvious in the case of the capillary vessels; in the larger trunks, as might be expected, not so invariably.

Of the vascular development, the following was noticeable; the enlargement of the postero-internal and external arteries, the occupation of the posterior median fissure by a considerable, sometimes very large vessel, and the presence of a branch of this also of fair size which penetrated Goll's column and traversed its midline from behind, forwards, through half its antero-posterior length. It was around this vessel that a definite excess of the neuroglia over the general hyperplasia was first seen. From this, it spread laterally, meeting a later developed growth around the postero-internal and septal arteries, until the whole column was invaded, whilst a similar increase was taking place in the area of the internal radicular fibres.

In one instance the middle third of both columns was the richest in connective tissue increase, apparently determined around the capillary terminations of the largely developed arteries running in from the cord periphery.

The posterior commissural zones were markedly involved in the neurogliar increase, and in two cases the latter appeared by its degree to have commenced there. Only in one was there a decided overgrowth of the pia round the cord.

The summary of a more attentive investigation than the foregoing outlines represent, seems to indicate a general increase of peritubular tissue and of vessels, as a stage precedent to any neural affection, and the localisation very frequently of the neurogliar excess around the arteries. Also an especial tendency to the involving of the posterior commissural zones.

It therefore seems to me that a special distribution in the one case, and a diffuse and impartial one in the other, is not to be taken as of much account in defining the systematic character of the tabes.

When research shall have settled the nature of the lesion in this disease as neural or perineural, an advance, and obviously a very important one, will have been made towards our knowledge of its etiology.

Before I had noticed Dr. Buzzard's papers in "BRAIN," in which he lays emphasis on the frequency of a vascular origin for tabes, the prominence of the vessel changes in the present case had not failed to arouse my attention, and their nature renders it impossible to disregard the conviction, that some very important part is played by them in connection with the development of the disease. It will be recalled that their distribution is in a large number of instances the centre for an increased growth of connective tissue, for the disintegration of the grey matter or cell-groups, that their coats show signs of inflammatory alterations, and constant indications exist of an active migration of lymph-cells and occasionally of the conversion of the latter into neuroglia-corpuscles. The condition of the investing membranes did not, however, lend any support to a theory of the origin in them of inflammatory action which might have spread in along the arteries.

Adamkiewicz has pleaded forcibly for a primary connective-tissue increase in tabes, determined around the course of the blood-vessels, and his statement lends strong support to Buzzard's views.

There is yet another pathological feature of interest occurring in a tract of close anatomical and functional relationship to the vessels and which demands especial notice, viz. the perivascular sheath and its contents. The changes in this are most obvious in the cord from the lower dorsal segment, upwards. In the regions below this the absence of the distended spaces is probably explicable by the universal sclerotic contraction, which not only obliterates the perivascular channel, but compresses the vessel walls almost to the rendering of the lumen impervious. It is likely also that obliteration of the perivascular space may occur by proliferation of the connective elements existing between its sheath and the adventitial tunic.

It is interesting to consider the relationship between the vascular and perivascular systems and the connective tissue overgrowth, and *à propos* of such, some few remarks may be made. Since the change is of the nature of an hypertrophy a condition of excessive nutrition is probable. All prolonged states of functional activity are associated with commensurate physical changes, and demand an increased supply of nutriment which is afforded by the blood plasma exuded from the capillary walls. It is recognised that since the capillaries run in lymph-spaces, the absorption of the plasma is not directly from the vessel walls but from the perivascular canal, as is also recognised the extremely intimate relationship between the lymph channels and connective tissue. An excess of exudation from the vessels under conditions when no call is made for it by the tissues, produces well-marked changes in the elements of the latter. Such undue exosmosis may arise from increased pressure in the arteries or from a morbid change in their walls; in the latter case, their permeability being increased, the escape of the morphological elements is much facilitated. And it is probable that migrated leucocytes are active agents in tissue proliferation. Supposing, now, that some system tract by increased functional activity demands those nutritive modifications which are needful to support a consequent increased tissue consumption, changes occur to secure a larger supply of nutrient fluid to the elements and this the latter derive in the manner before described. Under normal conditions such a freer supply when afforded is regulated by the demand, an equal balance being preserved between them, and an equable relationship maintained between the amount supplied and the effete products removed.

Some prominent causes of a disturbance in the harmony of this process at once present themselves. One may be, a condition of the vessels, general or local, inherited or acquired, causing a tendency to the occurrence in them of morbid changes. Should this exist, and circumstances be added which involve a constitutional or local impairment of nutrition, and others which require increased functional performance, disease of one or another kind of the vessel structure is likely to be developed, and one of the conditions needful for the readjustment of altered relationships is lost.

Another may be that predisposition, to which the term dyscrasia is applied, to a ready proliferation of tissue-elements, and perhaps also to the production of a specific form of effete material.

Thirdly, some state of the lymph paths, primary or derived, which by leading to vitiation of their contents, and by the blocking of their channels, to the stemming back of effete material, tends to produce a spreading of lesion along their course and to impair the vitality of the tissues which they drain.

In this way may, crudely enough, be carried back the pathological process to an origin, the exciting agent or agencies of which are yet to be found.

Concluding this portion of the subject, we are compelled to notice the pathological lesions in the brain in connection with the preceding remarks. Recalling what has been said regarding the dependence of the connective tissue hypertrophy on disturbance of nutrition and of the balance between the vascular and lymphatic systems, the presence in the brain of certain conditions of an assimilable kind to those in the cord, suggest again that the cerebral and spinal lesions are part of a wide-spread affection of the nutrient canals. The hypertrophic changes in Deiter's cells are known particularly to depend on conditions of lymph obstruction.

With reference to the peripheral zone of degeneration which is present throughout the lumbar region, this has been treated of by Déjerine and Westphal more especially, and who are exponents of different theories of its origin. The views of the former, ascribing it to an extension from a meningitis, itself set up by the posterior sclerosis, hardly suffice for the present case, since the membranes in the lower portion of the cord where the degeneration existed in the most advanced degree, were not markedly affected, whilst in the lower cervical, where the sclerosis was much less intense they were decidedly increased both in amount and vascularity, and the marginal lesion was absent. If, as Westphal suggests, it is of the nature of a system disease, since fibres in the cord presumably undergo degeneration throughout their length, such a tract would appear to be composed of fibres whose course is interrupted. Its position in the cord negatives its identity with the cerebellar tract. With regard however to that part of it in contact with the horn, it may be recalled that the posterior portion of the lateral column originates in germ-mass identical with that of the posterior column, and that therefore its participation in a degeneration of that region is not unexpected.

Various forms of intellectual and moral aberration are met with in connection with Tabes Dorsalis, and the question of their relationship to the latter disease is a most interesting one, unfortunately better recognised than understood.

Of all varieties of insanity, general paralysis has especially suffered an association. perhaps sometimes a forced one, with this spinal disease; not alone from the quasi-tabic symptoms so often existing in general paralysis, but from the tendency so constantly manifested in the two affections to approach in the bulb.

What the connection between the cerebral and spinal disease may be, is still under discussion, but that it exists is doubtless. The frequent coexistence of degeneration not only of centrifugal but of centripetal paths in the cord occurring in general paralysis, the, rarely, observed sequence of the latter to an apparently primary and independent spinal posterior sclerosis, and the by no means rare combination of a very constant variety of mental symptoms with tabes, all offer evidence of it.

That the disease spreads as a progressive invasive lesion along functional tracts is not however apparent. Spinal disease occurs, or at least manifests itself, secondarily to the cerebral in the majority of cases, and it does not suggest itself as travelling along centripetal tracts by continuity, in fact the knowledge of physiological laws here forbids its consideration. Moreover the earliest clinical symptoms, so far as objective signs are evidence, are not infrequently those resulting from the affection of certain bulbar nuclei, in tabes and general paralysis or of the optic nerve, and the representation of this nerve in the same portion of the hemisphere as the posterior columns of the cord and its intimate connection with the oculomotor nucleus renders its early involvement very interesting.

Returning to the bulbar lesion it is not obvious why certain nuclei should be picked out by a degenerative process, but that it is far from a haphazard event is certain. It seems however that certain physiological groups may have an especial inherent tendency to degeneration before others, and that where an unstable condition of the nervous system exists generally, or where actual lesion in some part of it is making progress, these will succumb, independent of any direct extension. The occurrence of a disease such as glosso-labio-pharyngeal paralysis which picks out a localised group for involvement tends to support this view.

It is not improbable therefore that in a conjoined cerebral and spinal affection, the respective lesions are not necessarily consecutive as regards anatomical continuity, but arise from a common cause, a proneness to degeneration of the nervous tissue at multiple points; an instance of which may be noted in cases of simple spinal disease. The development of the lesion primarily in the lower end of the cord, has its origin, it is likely, in some fairly definite source of extrinsic irritation; that of a cerebral lesion secondary to a spinal one, suggests a possible source of excitation in the latter, which some would indicate to be by the meninges, others by the vascular system, etc. And it is to be confessed that, when a progressive upward increase in the vascularity of the cord is apparent, together with a similar vessel development in the pons and medulla, and the vaso-motor phenomena which form a part of the symptomatology of both tabes and general paralysis are considered, the estimation of this latter view is enhanced.

Whatever be the lesion, its progress, or the department of the cerebro-spinal axis in which it starts, the theory that its predisposition is in an existing or inherent instability of the latter, and its excitation is, of functional origin, appears to be one full of strong recommendation.

The nervous system shaped and modified in its development by outgoing and all the ingoing stimuli constituting its environment, whilst dependent on them for its increasing complexity must be self reliant alone for stability. The part of the organism both structurally and functionally the highest evolved, it is that which unless possessing a proportionate vitality or stamina is like to suffer, first, dissolution. And as with the whole, so with its parts; and a functional tract which receives the most intense and complex stimuli will fall under the same law. It is obvious that as emotional expressions like pleasure and pain are largely determined in degree by the condition of reactionary power in the subject, so a long-continued or oft-repeated stimulus may become of physiological or pathological, developmental or retrograde, influence according to stability or the reverse of the nervous constitution.

The influence of heredity in the case of disease in any portion of the nervous system cannot be over-estimated. We can but faintly appreciate the transmission of the minutest details of tissue organisation from the parent to the offspring; of modes of mind engendered thus, and dependent on conditions and structural peculiarities of nerve matter, we can have hardly any conception. Certain paths are prepared, the action of certain mechanisms of nerve display facilitated, and the receptive organisation is modified for the more ready appreciation of special impressions: in such terms may we express predisposition. We may go so far as to denote the latter, as a shaping of portions of the organ to the necessity of some particular pabulum; for the food of nerve-tissue as regards functional development is stimulus; without that there is retrogression. Just as nerve mechanisms of special sense, auditory and optic, for example, may be adapted for complex combinations of sound and colours, so other parts of the receptive organ may be developed to the more ready imbibition of certain impressions. And in a faulty constitution of nerve-tissue, whose abstract correlative is manifested in all delicate gradations of the want of control and all the recalled states constituting judgment, are the conditions which render certain objective stimuli more powerful, and subjective reaction less commensurate. Such inherited defect of organisation manifests itself in some especially defined tendency, or in a general predisposition to yield to any predominant stimulus. This latter condition is perhaps the more common and the less defined ; it shows itself in feebleness in controlling the repetition of certain modes of nerve excitation, either determined by the physiological epochs of life, or chance circumstances.

When the intimate interweaving of the sexual function with the development of the organism is considered, its strong influence manifested in both sexes at certain periods of life, the intensity and variety of the sensations it gives rise to, and which are expressions of a very powerful and acute excitation of certain functional tracts, it is not possible to exclude it from a very marked causal influence in disease, an influence which, regarding the diffuseness of the natural sensations occasioned by the orgasm, the implication of the intellect and emotions for the time being, must involve the whole nervous system in the effects arising from its abuse.

There is a reticence so constantly observed in regard to the sexual relationships on the part of those from whom reliable information can alone be obtained, that statistics as to excess in this respect are perhaps doubtfully reliable. And whilst on the one hand tabes occurs frequently in men at the time when the sexual powers are vigorous and even verging towards the involutory period, yet in a class of women whose mode of life exposes them to a very constant indulgence of the animal passions, tabes is not prevalent. But this is by no means a more weighty evidence against sexual excess as influencing the production of tabes than it would be in the case of the closely related disease, general paralysis; for we must admit that the occurrence of a special disease more frequently in persons of one sex indicates something beyond a mere difference in degree of exposure to exciting causes.

The influence of heredity in our patient is sufficiently marked. As related in the history, the father was a most intemperate man, subject to paroxysmal attacks of passion; the mother, an asylum inmate suffering from chronic melancholia.

This heredity is apparent in the very dissolute habits of his life during adolescence, and a proneness to affections of both arterial and nervous structures may be considered inherent. For in the children of drunkards are frequent organic imperfections, early arrests of intellectual and moral development, and predisposition to inflammatory lesions; in them is especially apparent undue precocity, soon succeeded by retrograde changes.

Passing on to the consideration of the mental symptoms, as already observed, those in connection with tabes may assume various forms. At times they closely resemble those formerly regarded as being typical of general paralysis, and might even in such cases claim to be ranked under this head, were it not for the absence of certain motor characteristics of the latter affection, and, what is quite as important for the verification of the diagnosis, of definite pathological lesions.

General paralysis is a disease essentially compounded of two factors, the psychical and motor, but exists in so many, now recognised, diverse forms of the first, combined severally with the characteristics of the second, that it is the last which really constitute the disease as general paralysis. Separate factors, psychic and motor, but essentially coexistent.

Dwelling on the first of these for a moment, that the moral and intellectual spheres are affected differently both in time and degree is noticeable, and though both more often suffer deterioration, yet occasionally either is involved to a predominating extent; to use a metaphor, forming the heaviest weight in the balance the beam of which represents the psychical characteristics. Hence it may be said, that on the circumstances which shape the development of some special variety of insanity, independent of the somatic element, rests the determination of the psychical portion of the disease. Alcohol, syphilis, constitutional influences, sexual excess, may thus be the modelling agents in many cases.

Though, as has been said, general paralysis demands the presence of both such factors, their development is by no means commensurate, and one may far outstrip the other in its course. In remissions is this so, apparently to the complete disappearance of the mental with maintenance or even progression of the motor. Such features have an especial association with cases in which heredity is marked. (*Mendel.*)

If we then may accept that a paramount moral or intellectual deterioration combined with certain characteristic motor anomalies and pathological lesions constitutes the disease general paralysis, we incline to class the present case as one of such a category. It is probable that but for the untimely end by convulsions, symptoms more in accordance with the usual course of the affection might have developed.

It is in no way, however, desirable to force, as there may be a tendency to do, a similarity between tabetic insanity and general paralysis. In fact a connection between the cord affection and the latter disease is perhaps better brought into view, by cases which present signs of an intermediate nature. Indeed it is not the question of the connection of the mental symptoms of tabes and general paralysis, but of the relationship existent between the cord and brain. For though the mental aspect of the first named disease has been mostly alluded to, it is by no means single as a spinal affection in having cerebral complications, and the importance of this circumstance in the estimation of certain of these as being lesions involving both encephalic and spinal systems is obvious. Paralysis agitans and miliary sclerosis are forms of spinal disease which may here be cited. In each, too, as in tabes, the variety of psychic symptoms observed most often, is that of melancholia with delusions of persecution and hallucinatory disturbances. Nor is the reason of this depressed type of mental symptomatology an obscure one. The emotional state of the individual, whether sane or insane, is very largely the sum of the impressions derived from extrinsic and intrinsic sources; and their results are determined by the capability of judgment and control which exists. When these fail, are lost those means which enable the sane man to allocate, more or less correctly, effects to causes, by experience or inference. Blood and tissue alterations of a morbid nature in the sane and insane alike, provided the receptiveness of impressions is not too far inert, exert influence, though confessedly variable, over the emotional being.

It is difficult to imagine any disease more likely than a chronic, incurable, and advancing spinal lesion to influence with depression the whole mood of the patient's life, and when in the wide spread of the affection, the intellect is involved, and rational interpretations of his ills become distorted into a belief in them as the effects of machinations by known or unknown persecutors, as the results of loathsome disease or other cause, it is little wonder that the mental features are as some observers have stated them often to be.

Possibly, too, this depressed type of symptoms points, to a certain extent, to an etiology, in sexual excess, alcohol, and syphilis. The two latter especially of all, tend to produce a vitiation of nerve-tissue and an intellectual and moral depravation, and it is interesting to note in the three diseases, general paralysis, alcohol, and syphilis, that a lesion which may be suggestive of return to a lower type, meningeal adhesion occurs. Concluding these considerations on the nature of the mental condition in this case, in support of the view of general paralysis as existing, it is to be mentioned, that by some authors the predominance of a depressed type of symptomatology is urged in this disease: and also that it is, though rare, by no means unknown at so early a period of life, as that of our patient.

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EXPLANATION OF THE PLATE.

Fig. 1, 2, 3.—Diseased vessels in upper dorsal, cervical and upper lumbar regions of cord.

Fig. 4.—Fibres of Stratum Zonale—(*a*) swollen tortuous axis.

Fig. 5.—Longitudinal section of Artery from Posterior Columns. Granular masses on the intima.

Fig. 6.—Section of part of the Postero-internal Artery in Cervical region.

Fig. 7.—Semi-diagrammatic sketch of the transverse section of an Artery in the Cervical segment, surrounded by large granular bodies.

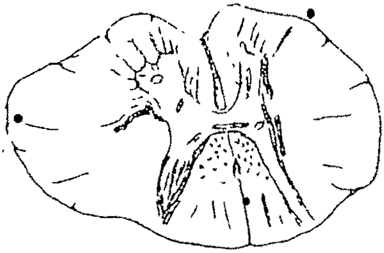

Fig 1

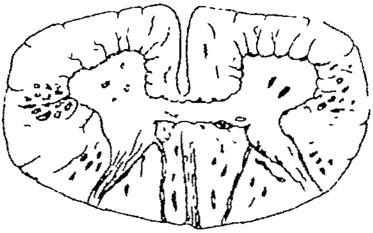

Fig. 2

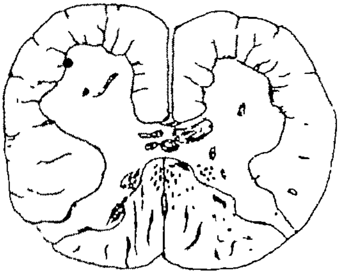

Fig. 3

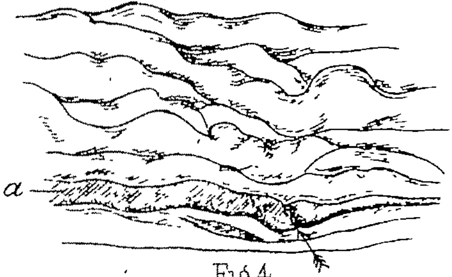

Fig 4

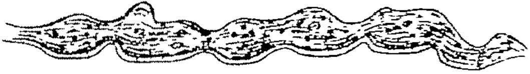

Fig 5.

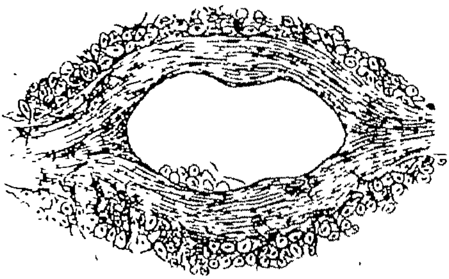

Fig 6

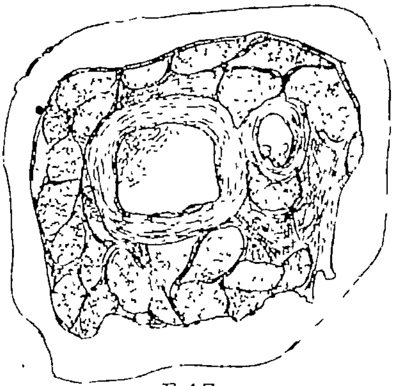

Fig 7

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PSEUDO-SCLEROSIS.

BY ROBERT MAGUIRE, M.D., M.R.C.P.

Physician to Out-Patients and Joint Lecturer on Pathology, St. Mary's Hospital.

IN March of 1887, Wm. B., aged 49, presented himself in my out-patient room. The patient was stout; the face was pale except for dark-coloured blotches on the cheeks. The skin had a greasy and unhealthy appearance; the hair was grey, but plentiful. The patient walked with an unsteady gait, the legs shaking as they were moved. When he was sitting up, the head and neck showed a continuous movement, which bowed the head downwards, and twisted the chin to the right, the occiput to the left. This movement, although continuous, was not regular in rhythm, or in extent. If the head were lightly supported, it almost, but not quite, disappeared; if the patient lay down so as to be entirely at rest, no movement was perceptible. The slightest attempt at voluntary movement of the head, or voluntary support of the head, or an attempt at speech, provoked an exaggerated and more rapid movement—always, however, of the same kind, and in the same direction. The lips were very tremulous when the patient spoke, but quiet at other times. The voice was shaky, the syllables were markedly scanned, pronounced slowly, and somewhat drawled; the words, however, were distinct, the pitch of the voice was monotonous. The hands and arms showed a general tremor even when the patient was at rest; afterwards, when the patient was admitted into hospital, it was noticed that a slight amount of this tremor persisted during sleep. The tremor was slow, not very fine, and not purposeful; the fingers had no special tremor, but moved with the whole hand, and even with the forearm. The tremor was greatly exaggerated when voluntary movement was attempted. The patient could not carry a cup to his mouth without spilling the contents; on attempting to do so, the involuntary movements, which were somewhat increased when the cup was held at arm's length, became more violent and a little more rapid, as the cup was brought nearer the mouth, until, when the cup almost touched the lips, the movement of the hand and of the head, which was brought down to meet it, were so great, that nearly the whole of the fluid in the cup was spilled before it could be got into the mouth. It was noted, however, that the movements were not at all choreic in character, and the hand was throughout carried in the desired direction, albeit in shaky fashion. The tremor was equally marked in both hands. When at rest, no tremor was observed in the legs; but when the patient attempted to rise, the muscles of the thigh were seen to shake, and the trunk was for a time unsteady; there was no sign of ataxia in the gait.

The grasp of the hands was feeble, and a general weakness was found on examining the principal muscles individually. There was no loss of sensation; there was no muscular atrophy.

The knee-jerks were greatly exaggerated; slight ankle-clonus could be elicited on the right, but not on the left side. The reflexes at the wrist and elbow were excessive, and jaw-reflex was also obtained. The superficial reflexes were normal.

The patient was somewhat lachrymose, but usually the expression of the face was apathetic; the mental state was childish. He complained of headache within an area about the size of the palm of the hand, at the top of the head. The pupils were dilated, and reacted very slowly both to light and accommodation; there was no nystagmus, no strabismus, and no diplopia. The ocular movements were carried out slowly, but this seemed to be from the stupid mental state rather than from inability to move the muscles. The right optic disc was white, the edges not well marked; but the blood-vessels were distinct; the left optic disc was irregular in form, but otherwise normal. No sign of optic neuritis was discernible; smell, taste, and hearing were normal.

The tongue was tremulous when protruded. It was red, scarred, bald and glossy in some places, covered with thick white flakes in others. The appetite was good, and thirst was complained of; the abdominal organs were normal. In the lungs, signs of emphysema were observed. The pulse was large, soft, regular and full, and beat 68 times in the minute.

There was some pain on passing urine, and the stream was small; the urine was amber-coloured, clear, sp. gr. 1028, faintly acid, contained neither albumen nor sugar, and deposited phosphates on standing.

The temperature continued normal throughout his stay in the hospital.

The patient was born in London, but had lived most of his life in other parts of the country, and served in India and the West Indies for a time as a soldier. He was never intemperate, and retired from the army after fifteen years' service with a small pension and several good-conduct badges. Latterly he had been very badly off, thinly clad, and short of food, and had slept out in the streets for three nights during the fortnight before admission. The reason of his poverty was that the tremor of his hands prevented him getting work.

The family history was of no importance. He had suffered from gonorrhœa and syphilis, the latter 22 years ago. He was attacked by yellow fever in the West Indies; after this his hair turned white, and has remained so. He did not think he was treated with mercury for the syphilis, and certainly was never salivated.

The patient first noticed that he had a twitching at the back of the neck, with sometimes trembling of the hands, in 1876 (when he was nearly forty years of age). It gradually became worse, until in 1878 the medical staff of the Royal Marine Artillery discharged him as unfit for service. The disease progressed, and the patient stated that first the neck and head, then the arms and then the hands, were affected. Only about five months before admission had the legs commenced to be affected, and caused him to walk unsteadily. He had at different times applied for treatment at various hospitals, and been more or less relieved.

The patient was kept at rest in bed, and was given iodide of potassium in 10-grain doses, with a draught of bromide of potassium and chloral hydrate at bedtime.

On March 30th he seemed much better. The tremors were of much less extent, the sleep quieter, and the headache less. On April 14th the step was quite steady, very little shaking of the head could be perceived, and the lips were no longer tremulous; the speech had lost its shaky character, but was still slow, monotonous, and somewhat scanned. No tremor of the hands was now observed when at rest, and but little when voluntary motion was attempted. The patient was able to feed himself and drink liquids without spilling them. The knee-jerks were lively, but there was no ankle clonus. The mental state was still childish, but the patient was no longer so emotional as when admitted. On April the 21st he went to a convalescent home for six weeks, and I heard from him whilst there to the effect that tremor had almost entirely left him.

I have since been informed that some time after leaving the Convalescent Home he was admitted into the Union Infirmary, with a return of symptoms resembling those for which he was treated in St. Mary's. This time, however, he was relieved to a certain extent without the use of iodide of potassium.

Naturally in this case the diagnosis was perplexing. One thought in the first place that the extreme hardships which the patient had had of late to undergo might have produced the symptoms. It is occasionally seen that simple malnutrition of the nervous centres from starvation will cause tremor, and excessive reflexes, but the history in this case was clear that the tremor had induced the hardships, and in consequence of it the patient had been dismissed from the army. Mercurial tremor may produce an assemblage of symptoms very like that described, but this cause was carefully eliminated. Although the case was lacking in many of the symptoms of disseminated sclerosis, and although some of the symptoms found are not met with in the latter disorder, yet this was the disease which was most forcibly suggested on examining the patient. The persistence of the tremor resembled that of paralysis agitans, but the tremor in this disorder is finer, purposeful, does not affect the head and neck muscles, and moreover does not show the exaggeration on voluntary movement described above. On the other hand, a diagnosis of disseminated sclerosis did not seem to be altogether permissible. As mentioned above, the symptomatology did not entirely correspond with that of disseminated sclerosis, but nevertheless in the latter disorder the symptoms in different cases are variable, within somewhat wide limits. The age of the patient when the first symptom appeared was very late for the commencement of disseminated sclerosis, and also we should have expected after twelve years a much more pronounced degree of the disorder at the time when he came under observation. Yet these are only probabilities. Disseminated sclerosis may, in rare instances, first show its symptoms at a very late period of life. Strümpell, for instance,[1] mentions that he has observed a case in a man 60 years of age, and confirmed his diagnosis by an autopsy. Again, disseminated sclerosis may sometimes pursue a very latent course. Nevertheless, balancing the probabilities of the whole case, I diagnosed, not disseminated sclerosis, but a functional disorder simulating it. This I think was confirmed by the progress of the case. It is difficult to believe that marked symptoms due to organic lesion could show such rapid amelioration as was here observed. In the earlier stages of disseminated sclerosis more or less improvement may be sometimes observed, but after twelve years' duration of the symptoms we could hardly consider any patient to be in an early stage. Syphilis we know may simulate the symptoms of general paralysis of the insane, and yet the recovery of the patient marks the difference between the two affections. Thinking of this I was at first inclined in the case reported to ascribe to syphilis the rôle of producing the symptoms, although, I believed, without the presence of a local lesion. For this, however, there was no real ground, and the second recovery, of which I have heard, without the use of iodide of potassium, would be opposed to such a view.

Westphal in 1883[2] described two cases in which during life many of the symptoms of disseminated cerebro-spinal sclerosis were present, and yet after death no lesion whatever was found. For purposes of comparison, I may be permitted to give a condensed abstract of the exhaustive reports on these two cases.

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[1] Specielle Pathologie und Therapie IIa., p. 189.

[2] Archiv f. Psychiatrie, xiv. p. 87.

The duration of the disease was in the first case nine, and in the second ten years from the appearance of the first symptoms. In both cases there was motor weakness of numerous muscles of the limbs and trunk, sometimes with spastic phenomena. In both cases the movements of the head were slow, tremulous, and interfered with by involuntary movements of the neck muscles. The gait was spastic-paralytic; the spastic affection was almost entirely confined to the muscles of the lower limbs; passive movements met with more or less resistance, and there was usually tremor on voluntary movement. In the upper extremities there was motor weakness, and also marked tremor on voluntary movement. In both cases the knee-jerks were present, and also Westphal's paradoxical contraction. Sensory symptoms were almost entirely absent in both cases, and the bladder and rectum were unaffected. In the first case the speech resembled that of bulbar paralysis in not being sharply articulated; but in the second case it was more scanning, without any affection of the sharpness of articulation; in both it was monotonous.

There was no nystagmus or paresis of the eye muscles, although in one case double vision had been complained of before the patient came under observation. The movements of the eyes were, however, very slow, and performed with difficulty. This symptom, Westphal remarks, is occasionally found in paralysis agitans, when the muscles become stiff, but has not been observed in multiple sclerosis. The expression of the face was stupid, the face-muscles in one of the cases trembled during speech.

Headache and dizziness were complained of, and in both cases there were psychic disorders. In the first case there was general intellectual weakness; in the second a childish manner and excessive irritability. In this latter case curious attacks occurred in which consciousness was lost, and hemiplegia of the right half of the body without affection of the facial muscles followed. Other paralytic phenomena of both sides with diminution of sensibility were observed later on.

The course of both cases was extremely chronic; the symptoms occasionally showing marked and rapid improvement. Death in the second case occurred suddenly in the night, and in the first resulted from a scorbutic condition.

At the autopsies, no change whatever, neither in the central nervous system, nor in the peripheric nerves, could be discovered in the body of either patient.

There are, it is clear, many points of difference between these cases reported by Westphal and my own, yet there are also many points of resemblance. All three presented many symptoms of disseminated sclerosis, and from a perusal of Westphal's paper, I take it that he did not wish to describe these two cases as illustrative types of a new disease. To quote the conclusion with which Westphal ends his paper: "There is a general neurosis, which one may possibly, if not very happily term pseudo-sclerosis, and which can be separated clinically from disseminated sclerosis, neither by its symptoms nor its course." I have therefore ventured to place my case in this category, seeing that it much resembles disseminated sclerosis in its symptoms, and yet, for reasons which I have already fully detailed, it seems to me probable that no organic lesion exists. Naturally, however, the criticism will be made—and, I admit, justly—that in the absence of an autopsy such a diagnosis is very uncertain.

ON SPEEDY RECOVERY FROM THE EFFECTS OF CEREBRAL EMBOLISM.

BY J. S. BRISTOWE, M.D., LL.D., F.R.S.

ABSOLUTE recovery from the effects of embolic blocking of any of the larger arteries of the brain is, for reasons which are well understood, scarcely to be hoped for in any case that comes under treatment, and must certainly be of very rare occurrence. With respect to the similar obstruction of any of the cerebral arterioles, which supply only minute districts, the conditions are different; inasmuch as, although these vessels are still without anastomoses, and serious, though limited disturbance of circulation must necessarily be the immediate consequence of the accident, it is possible that the circulation may be restored and maintained efficiently, through capillary connection with adjoining arterial districts.

The cases which follow exemplify, I believe, both of these occurrences.

In the first case, the patient, who had been under my care on and off for years with heart-disease, the result of rheumatism, was attacked suddenly with the usual symptoms of obstruction of the left middle cerebral artery, namely right hemiplegia and aphasia. Looking to the facts, there could be no reasonable doubt that her paralysis was due to embolism; and the medical man who was called in took this view, and formed an unfavourable prognosis. But she recovered perfectly in the course of a few hours, and remained free from all trace of paralysis for over four months; at the end of which time she had another attack, presumably due to re-obstruction of the same artery, which left her permanently crippled. I do not see how anything save an embolus can have caused her first seizure; and the only suggestion I can offer to explain her recovery is that the embolus was a loose friable mass, which, after it became lodged, broke down into minute fragments, and was thus dispersed.

CASE 1.—*Heart-disease from rheumatism: followed by temporary right-sided hemiplegia and aphasia, and four months later by permanent right-sided hemiplegia with temporary aphasia.*

A. L., a married woman, aged 25, was admitted on the 11th of January, 1887. She first had rheumatism some seven years ago; and since then has been several times in the hospital under my care for heart-disease, and has frequently also come to see me as an out-patient. During all this period she has had a double mitral murmur: and from time to time has suffered from dropsy and hæmoptysis, from which, with rest, she has each time recovered.

About four months ago she came to me, apparently in her ordinary state of health, and suffering neither from dropsy nor from spitting of blood, to get my advice in reference to some symptoms which she had lately experienced. The following is the account she gave of herself. About a fortnight previously, her husband, whose occupation compels him to get up very early in the morning, went to his work as usual at about four o'clock, leaving her in bed. Between seven and eight o'clock she rose, feeling fairly well; but almost immediately afterwards fell down, paralysed on the right side and unable to speak. She lay helpless on the floor for half-an-hour or more, until the landlady of the house in which she lodged, attracted by her moaning, came to the room. A medical man was at once fetched, who recognised that she had right-sided hemiplegia with aphasia, assumed it was due to embolism, and foretold an unfavourable issue. The right arm and leg were absolutely powerless, the mouth was drawn to the left, and she was quite unable to utter any articulate sound. But she retained her consciousness. The symptoms continued without change for three or four hours, and then subsided. And on the occasion of her visit to me there remained no trace whatever of paralysis, her speech was perfect, and she was (as stated above) apparently in her usual state of health. The medical man referred to subsequently, in a letter, confirmed the substantial truth of the above narrative.

She continued in her usual health until a week before admission; when, on waking in the morning, she again found herself paralysed on the right side, and unable to speak. During this week her speech became almost perfectly restored; but the paralysis underwent no change.

State on admission.—A spare, interesting-looking young woman; suffering from well-marked right hemiplegia, without loss of feeling. The right arm and leg were absolutely motionless; the mouth was symmetrical when at rest, but drawn to the left when showing her teeth or smiling; the tongue was protruded to the right; and the right palpebral fissure was usually a little more open than the left. The tendon and superficial reflexes were natural. Her articulation was not perfect; and she was a little slow at speaking, as if her words did not come quite readily. Moreover, there was an occasional slip in naming things; thus she called an ink-stand an ink-pot or ink-can. For the most part, however, her words were correctly applied. The heart's apex beat in the sixth space, an inch outside the nipple line. Its action was somewhat irregular. There was a loud, harsh, systolic, and a well-marked præsystolic murmur, chiefly audible at the apex. In all other respects she was fairly well.

She remained in the hospital until the 17th of April; and during her stay there was up to a certain point marked improvement in her cerebral symptoms. For a few weeks she still presented slight impairment of articulation, and unreadiness in naming; but during the latter part of the time her speech became perfect. Moreover she could read: and was able (though with awkward formation of letters) to write with her left hand. The slight facial and lingual paralysis gradually disappeared. Her leg improved slowly, and before she left the hospital she could pretty readily flex and extend the hip, knee and ankle joints, but still could not move her toes, or walk. She did not regain any use of the right fingers, hand or forearm; but to some extent recovered the power of moving her arm at the shoulder joint. After she had been in the hospital about a week, the tendon-reflexes in the right arm and leg had become exaggerated with development of ankle-clonus, and the superficial reflexes had diminished; and these phenomena continued thenceforth. It may be added that on several occasions it was noticed that when the patient yawned, the right hand and its third and fourth fingers became involuntarily extended.

Occasionally during her sojourn in the hospital she suffered from symptoms referrible to the heart; and occasionally also abnormal cardiac sounds were heard which, it was thought, pointed to affection of the tricuspid valve. But no other incident calling for notice occurred.

The second case was that of a woman who was under Dr. Stone's care, and whom I had the opportunity of examining from time to time. She was admitted with mitral-valve disease of old standing; and while under observation in the hospital had sudden obstruction of the abdominal aorta and left radial artery. She presented the characteristic phenomena and consequences of such obstructions, but so far recovered as to be able after a time to leave the hospital, still with incomplete restoration of the affected limbs, yet feeling on the whole fairly well. There can be little doubt that these sudden arterial obstructions were embolic. The case is chiefly interesting for the reasons above given. I quote it, however, on other grounds. Eight or nine days before she was discharged, and at a time when she had in great measure recovered from her more serious symptoms, she was attacked with paralysis of the left internal rectus, of the right facial nerve, and of the right external and internal recti, with dilatation of the right pupil, vomiting and fall of temperature. The fundi of the eye remained normal. Having regard to the facts, that the patient had heart-disease of rheumatic origin, and that she had recently had sudden obstruction of the aorta and of the radial, it is scarcely possible to doubt, that this last attack of paralysis was due to a minute cardiac embolus causing obstruction of some small artery, distributed to a small district of the right crus cerebri and adjoining pons. In the course of a few days all the paralytic and other symptoms due to the accident had disappeared absolutely.

CASE 2.—*Mitral disease—Sudden obstruction of aorta with disappearance of pulsation from abdominal aorta, and arteries of both lower extremities, and of left forearm—Paralysis of legs: temporary suppression of urine—Partial recovery; followed by paralysis of certain muscles of eyes and right facial nerve, of short duration.*

C. M., a married woman, aged 33, was admitted under Dr. Stone's care, on the 4th of February, 1883.

She had rheumatic fever seventeen years previously, but never any second attack; she had five children, of which two have died, and the youngest is 9 years old; has been in poor health for the last eight or nine years; and has suffered latterly from dyspnœa, cough, and pain in the region of the heart.

It was for these symptoms she was admitted. And, on examination, the heart seemed little if at all enlarged or displaced; but there was a distinct præsystolic thrill, and murmur at the apex; there was no dropsy, or albuminuria, and only a little crepitation at the bases of the lungs.

During the early part of the 6th, she had some numbness in the feet, and in the afternoon complained of severe pain about the heart, and faintness. About 5 P.M. she had a sudden pain below the left ribs, and vomited immediately afterwards.

The next day the pain and faintness and sickness had ceased; but the feet were cold; there was no pulsation in the abdominal aorta or in the main arteries of the lower extremities; she could flex the knees, but could not move either the ankles or the toes; there was loss of sensation from the knees downwards; the superficial reflexes were absent, and the patellar tendon-reflex was deficient in the right lower limb, where also there was a burning gnawing sensation. Later in the day, it was discovered that there was no pulsation in the left radial. She was anxious-looking;

her pulse was 80, full; and the heart's sounds remained as before. The temperature rose to 100·2.

After this, the condition of the patient underwent very little change for some time, but on the whole improved decidedly. The heart's action was not rapid, but was inclined to be irregular; the legs remained weak, but she recovered some power over the movements of her toes, not of her ankles; sensation returned to a considerable extent, but there still remained some numbness of the feet; the patellar tendon-reflex remained absent from the right leg; no pulsation was ever detected in the abdominal aorta, but it was fancied from time to time that it could be felt in the arteries of the lower limbs, and in those of the left forearm; some superficial patches of gangrene appeared on both legs, but remained of small size. It may be added that, for a day or two after the onset of severe symptoms, the urine was almost entirely suppressed, and she suffered from drowsiness.

At the end of March her condition was much better than could have been hoped for. She looked well, was free from pain, and cheerful; and she had recovered to a considerable extent the use of her legs and left arm; but the former were still weak, the pulsation in the formerly obstructed vessels was very feeble and often undiscernible, and the cardiac sounds and action were in the same state as when she was admitted.

On the 31st of March, however, the patient felt ill; and on the 1st of April she complained of diplopia. The right pupil was dilated; there was weakness of both external recti, of right internal rectus, and also of right facial. She had vomited once; and her temperature had sunk to 95.

On the 2nd the pupils were equal, but the diplopia continued, the weakness now being apparently limited to the right internal rectus, the left external rectus, and the right facial (which had diminished). She vomited frequently during the day. Her temperature remained low. After this, the paralytic symptoms about the face gradually disappeared; the sickness subsided; and she returned to the condition in which she was prior to the 30th of March. The eyes, examined by the ophthalmoscope, were said to be healthy; her temperature remained subnormal.

She left the hospital at her own request, on the 7th of April; the circulation in the affected limbs being still imperfect, her power over her legs still incomplete, and her ability to walk still impaired.

The next case I take to be of the same kind as the one last narrated. But the symptoms were milder and the evidence of embolism less clear.

CASE 3.—*Temporary tendency to cross paralysis of the face, due probably to embolism.*

A young medical man, who had had two attacks of acute rheumatism, of which the last had occurred eight years previously, and who had suffered ever since he was a child from megrim, consulted me about himself in November, 1879. With the exceptions above specified he had generally enjoyed good health, and had never had syphilis. But for three or four months he had been somewhat out of sorts, and had slept badly. The day before he called upon me, while sitting in his chair, he experienced a strong sensation of warmth, with tension, on the right side of the face. He at once got up, dipped his face in water; and then, on looking at himself in the glass, discovered that he was very pale, that his sight was confused, that he had slight double vision (with, as his account seemed to show, some obliquity), and that his left pupil was dilated to the full, and inactive to light and accommodation. He was a little tottery in his walk. But he was quite himself, had no sensory disturbances, was wholly free from paralytic weakness of face or limbs, and was not aphasic. Headache referrible to the left side of the head followed. These symptoms continued during the remainder of the day; he passed a restless night; and the following morning he found that his pupils were equal and natural, and his squint was gone. When I saw him in the afternoon he still felt a little shaky; but all the other special symptoms had subsided. On examination of the heart, I detected a very slight systolic murmur audible at the apex, only at the end of a deep inspiration, and inaudible behind. I concluded that the heart was mechanically healthy; but suspected from his history and recent attack that there were a few granulations on the auricular aspect of the mitral valve. The patient is still in good health. The sensory affection of the right side of the face, combined with the dilatation of the left pupil and double vision, led me to conclude that the lesion causing his symptoms was in the neighbourhood of the pons. And from the rheumatic history, and temporary character of the attack, I attributed it to a small embolus lodged in one of the vessels of this part. The case at any rate has a close resemblance to the last. I need scarcely add that there must have been paralysis of one or more of the ocular muscles, but of which there was no evidence.

Clinical Cases.

MULTIPLE PARALYSIS OF CRANIAL NERVES—DEATH—TUMOUR OF PONS AND CEREBELLUM.

BY THOMAS BUZZARD, M.D., F.R.C.P.

Physician to the National Hospital for the Paralysed and Epileptic.

EDWARD L., æt. 40, was admitted into the National Hospital for the Paralysed and Epileptic, Queen Square, on August 31st, 1887, suffering from paralysis of various cranial nerves. Previous to admission, patient had been under the care of Dr. Ormerod.

Family and personal history unimportant. The patient had never suffered from syphilis. The following account is derived, in large measure, from notes taken by Dr. Williamson, Junior Resident Medical Officer to the Hospital.

There is paralysis of the sixth, portio dura, and portio mollis of the right side, with a little facial anæsthesia and slight weakness of right pterygoid muscles; whilst the arch of the soft palate is much reduced on the same side, the uvula deviating to the left. The right half of the palate remains motionless when patient says "Ah!"

The patient has a staggering gait. At first it seemed that this might be the result of erroneous projection due to the paralysis of the external rectus muscle of the right eye, because he walked much more steadily when this eye was shut; but latterly this has not been the case, and he has staggered, whether one eye or the other, or both were open. He complains of giddiness only when walking. He does not, however, complain of much weakness in the limbs, except as regards the legs. But it is very noteworthy that the tendon reflexes, both at the wrist and knee, on both sides of the body are enormously increased.

It seems that two years ago patient was attacked with headache in the morning, chiefly in the frontal region; and about six months later began to see double, and his wife noticed that his face was distorted. Twelve months ago he became deaf on the right side. The unsteadiness of gait has been observed for about six months. The face is paralysed on the right side, but, owing to secondary contraction of muscles, there is a *primâ facie* aspect of left-sided paralysis, the naso-labial fold being well marked on the right and absent on the left. He cannot quite close the right eye. The muscles of the right side of face react very feebly to induced currents of moderate strength, which produce powerful contractions of those of the left side, and with a galvanic current of 5 m.a. on the right side, A S Z > K S Z.

The tongue is protruded straight. There is nothing abnormal in the condition of the vocal cords as seen by the laryngoscope.

When the patient opens his mouth, the jaw is carried a little over to his right, owing apparently to the pterygoid muscles of the right side being somewhat weaker than those of the left. It is thought, too, that the lateral movement of the jaw is stronger towards the right than the left side, and when patient closes his jaw tightly, the left masseter is more prominent than the right.

The sternomastoid and border of the trapezius may possibly be a little better marked on the left side of the neck than on the right. To induced currents of moderate strength both react equally well.

Patient can feel the touch of a feather a little better on the left side of tongue, palate, and inside of cheek than on the right, but on the lips the touch is perceived equally well on the two sides.

The deafness on the right side is absolute, and considered by Mr. Cumberbatch, who examined him, to be of central origin.

Quinine and salt-solution are perceived less distinctly on the right than the left half of the tongue.

The condition of the viscera presents no abnormality.

Under treatment with iodide of potassium and mercury the patient to a great extent lost his frontal headache, but the other symptoms remained unaltered, and in addition he began to complain of some difficulty in swallowing, the food, as he described it, tending to go to the right.

In December the patient became somewhat rapidly worse. The difficulty in swallowing either solids or fluids increased. Food would often pass into the glottis and set up coughing. On December 27th it was noted that he was unable to walk without assistance, owing to weakness of his legs, and there was a marked tendency to fall to the right, whilst the difficulty of swallowing became still worse.

On January 2nd it was noted that the pupils were equal in size, and that there was no optic neuritis.

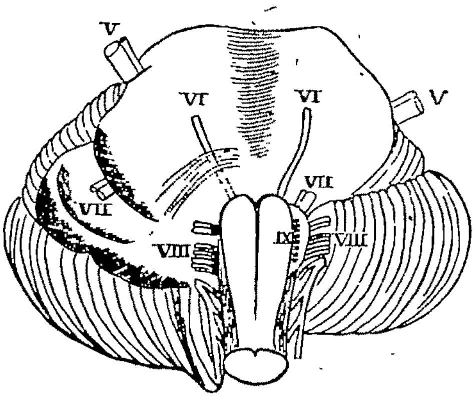
On January 4th the patient was unable to stand, and only able to raise the legs a few inches from the bed in the extended position, but could flex and extend the feet fairly well. He was also able to flex the hip and knee-joints, though feebly. The head of a pin was distinguished on the outer side of the legs and on the dorsum of the foot, but on the inner side of the legs sometimes it was not felt. A pin prick was felt as a prick in both legs. The plantar reflexes were very active on each side—and so were the knee-jerks. There was no ankle-clonus. The head of a pin was felt on both upper limbs, but rather better on the left side than on the right. He could only take liquid food. The tongue was protruded to the right. There was occasional vertical headache, but much less severe than it had been: no vomiting.

The mental state seemed dull, and he was slow in answering questions.

At the end of his dinner he suddenly fell asleep, slept heavily, and about 3 P.M. his breathing became stertorous and face livid, the skin covered with cold perspiration. His pulse was 120, fairly strong and regular. The temperature 102; pupils contracted, conjunctivæ insensitive. He could not be roused, and died shortly afterwards in a state of coma.

On post-mortem examination a new growth was found at the base of the brain. It could be separated into two halves of about equal size, the two portions being in very close apposition, and only separated by a narrow slit.

The inner portion occupied the surface of the inferior part of the right half of the pons. It measured about one inch by three-fifths of an inch. The colour was perhaps a little darker than the rest of the surface of the pons, although it bore a very strong resemblance to the rest of the brain surface. Fewer blood-vessels ramified upon it than upon the brain surface generally.



V, the trifacial nerve; VI, the abducent ocular; VII, the facial and auditory nerves; VIII, the glosso-pharyngeal, pneumo-gastric, and spinal accessory nerves; IX, the hypoglossal nerve.

Occupying the under and anterior surface of the right lobe of the cerebellum (and immediately adjacent to the just-described growth upon the pons) was a mass of precisely similar appearance, about 1½ inch in longest diameter, and ¾ inch across. These two pieces were in close contact, only a thin slit separating them. Through the middle of this slit the right facial and auditory nerves projected, being displaced and evidently compressed by the two halves of the growth. The superficial origin of the right sixth nerve was just in front of the outer and upper angle of the growth, but not adherent to it. The posterior part of the outer (cerebellar) mass was in contact with and adherent to the eighth nerve (glossopharyngeal, vagus, and spinal accessory), but the spinal accessory portion did not appear to be involved in the growth, which did not reach down to its superficial origin.

The medulla oblongata was pushed over to the left; the olivary body on the left side being very prominent and somewhat hard to the touch, but the appearance of the surface was normal. The olivary body of the right side appeared much smaller than that of the left, apparently from the compression to which it had been subjected.

The growth upon the cerebellum concealed the flocculus, and appeared to displace the amygdala and biventral lobe of the right side far downwards. It pressed upon the middle cerebellar peduncle of that side.

The middle lobe of the cerebellum was far out of reach of the growth, and could not apparently be affected by it, even indirectly.

This patient was exhibited to the Neurological Society on November 10th, 1887. At that time no inconsiderable doubt was felt as to the situation of the lesion, and I drew attention to the following points.

The well-marked paralysis of the facial muscles, involving as it did those of the upper as well as the lower half of the face, pointed to a lesion of the pons rather than one of the bulb, and the paralysis of the fifth and sixth nerves would also accord with a lesion in the former situation. On the other hand, what difficulty there was in swallowing at that time was not at all of a strongly marked character, and there was no such impairment of articulation as might have been expected if the lesion were in the pons. Neither was there any paralysis of limbs, and the sensation was undisturbed. The unilateral paralysis of the palate again appeared to indicate the bulb rather than the pons as the seat of lesion. The fact of the portio mollis being implicated along with the portio dura would point to lesion of nerves rather than of centre, and so would the paralysis of the sixth unaccompanied by impairment of the third. A tumour, or analogous coarse lesion, involving the middle fossa of the skull on the right side, might explain most of the symptoms; indeed, one's first thought in such a case was of a syphilitic gummatous formation in the floor of the skull. It was easy to imagine the meatus auditorius internus encroached upon, so that both the portio dura and mollis which lie in it should be subjected to destructive influence. The large superficial petrosal branch of the Vidian nerve lying in a groove on the petrous portion of the temporal bone would easily be involved in a lesion occupying this situation. If the view were taken that the nerve supply to the levator palatæ and azygos uvulæ was derived ultimately from the portio dura, through the superficial petrosal branch of the Vidian connecting this nerve above its geniculate ganglion, with the spheno-palatine ganglion, the right-side paralysis of the soft palate was at once explained. Again; the close proximity of the Gasserian ganglion and its possible implication would account for the disturbance of sensibility in the right half of the face and mouth. There was more difficulty in explaining the affection of the sixth nerve, which is separated by the trunk of the fifth from the portio dura and mollis. It seemed hardly likely that a tumour in the middle fossa, which involved so gravely the sixth and seventh nerves, would only affect to so slight an extent the trunk of the fifth lying between them.

In these circumstances I did not feel able to express any strong opinion as to the position of the lesion, whether it were one of the floor of the skull involving the affected nerves, or a growth in the pons or bulb influencing the nuclei. The remarkable exaggeration of the tendon reflexes, however, made me incline to the latter opinion, as it seemed probable that this was due to pressure upon the pyramidal tracts.

A few weeks later the increase of difficulty in swallowing, and the growing weakness of the patient's limbs, made it unlikely that the lesion was in the floor of the skull, and the symptoms, taken altogether, appeared so conclusive, that I felt able, before the autopsy, to express the opinion that in all probability a tumour of the Pons Varolii would be found influencing the cerebellum by pressure.

The result of the autopsy leaves still undecided the vexed question of the mode of innervation of the levator palati. The facial nerve was compressed and paralysed, it is true. On the other hand, although the superficial origin of the spinal accessory appears to have escaped the encroachment of the growth, yet the medulla oblongata was pressed upon by the tumour over the whole of the olivary body. If a horizontal section be made through the bulb towards the lowest part of the olivary body, it cuts through the bulbar origin of the spinal accessory as well as the commencement of the hypoglossal nucleus. From the fact of the twist of the tongue to the right in the last part of this patient's illness, it is evident that there was paralysis of the hypoglossal on the right side. This must have been through lesion of the nucleus and not of the trunk of the nerve, for this was out of reach of the growth. *A fortiori*, it is probable that the nucleus of the spinal accessory above it was likewise subjected to damaging pressure. If the levator palati be innervated from the spinal accessory nerve, its paralysis might possibly thus receive explanation. It is right, at the same time, to remark that the vocal cords were unaffected, and that the sterno-mastoid and trapezius of the same side, although possibly, but not certainly somewhat less developed than those of the left side, showed no alteration whatever in their reaction to electrical currents. On the whole, therefore, considering the severe compression to which the portio dura was certainly subjected, and which had produced a typical paralysis of facial muscles, the evidence, such as it is, would point rather to the portio dura than to the spinal accessory as the source of innervation of the levator palati.

Microscopical examination showed the growth to be a round-celled sarcoma. It had apparently started from two points of origin, one on the pons, the other on the cerebellum, and these in their growth had met and moulded themselves against each other, squeezing the portio dura and portio mollis between them, whilst the trunk of the fifth nerve was pressed upon and displaced upwards and forwards.

A CASE OF DOUBLE WRIST DROP APPARENTLY DUE TO MULTIPLE NEURITIS OF ALCOHOLIC ORIGIN, THE LOWER EXTREMITIES HAVING PERFECTLY RECOVERED.

BY THOMAS BUZZARD, M.D., F.R.C.P.

Physician to the National Hospital for the Paralysed and Epileptic.

JAMES M., æt. 42, engine-fitter, was admitted into the National Hospital for the Paralysed and Epileptic on January 31st, 1888, with loss of power in both hands. The following account is derived from notes taken by Dr. Williamson, Junior Resident Medical Officer.

The patient, a fairly well-nourished man, with puffiness below the lower eyelids, described how, on March 24th, 1887, he found on awaking in the morning that he was unable to use his hands. He had used them freely the day previously. The fingers were semiflexed, and patient was unable to extend them, nor could he bend his elbow. He was unable to get his hand to his mouth. He was just able to stand but not to walk. His hands felt numbed, and he could not feel when anything touched them, nor could he hold anything with them.

There was no affection of speech, no pain in the head or back. He noticed then for the first time that his arms looked thin.

He was unable to walk without help for two or three months.

He did not feel any numbness in the lower extremities. When he commenced walking he could not lift his feet, but dragged them.

The attack took place when he was on board ship off Rio Janeiro. There was no other case of illness on board, and he had not been on shore.

The hands have been gradually getting worse, and patient has been unable to do any work since the onset of paralysis. The arms, he says, have been gradually getting thinner. He had never felt any pains or rheumatism in his arms.

As regards antecedents, he had suffered from scalp wound and erysipelas of the head and face two years ago, and thinks he was unconscious for four weeks. He has had ague four times slightly. There is no specific history. He has had gout in the right big toe three times. There is no history of acute renal mischief. Many years ago he used lead in fitting joints of machinery, but not for the last six or seven years. In his trying work in the engine-rooms of steamers in the tropics he has drunk very freely indeed. He had been taking about fourteen ounces of whisky daily, besides malt liquor for two or three years at least before his attack.

The grasp of the right hand is 25, that of the left 30. Both hands are dropped at the wrist, and the fingers semi-flexed without contraction. There is slight wasting of the thenar and hypothenar eminences, more on the right hand than on the left, and of the first interosseous space on the right side. Patient is unable to fully extend the fingers. He is able to separate and approximate the fingers of both hands, but not to the full extent.

He can only imperfectly extend the wrists, and flexion is feeble. All movements of the elbow and shoulder can be performed. He can pronate and supinate the hand. There is slight wasting of the muscles on the extensor side of the forearm.

The other muscles of the forearm are somewhat flabby, but not wasted. The supinator longus stands out on each side, when the arm is flexed at the elbow, the forearm being in a position between pronation and supination.

The wrist-jerk and triceps-jerk are present on both sides.

There is nothing wrong to be observed about the spine or lower limbs.

The patient walks well, without unsteadiness. The knee-jerks and plantar reflex are normal on both sides. There is no ankle-clonus.

There is no affection of the facial or ocular muscles, and the tongue is protruded straight. The pupils react to light and to accommodation. Symptoms of commencing albuminuric retinitis are observable in the left eye.

There is no affection of cutaneous sensibility.

No blue line is to be found on the gums. The circulatory and respiratory systems show nothing abnormal. The urine is sp. gr. 1012, alkaline. It contains albumen—no sugar. No casts are observable. The patient micturates very frequently.

Electrical examination; Galvanism.—In the forearms all muscles react readily except the extensor communis digitorum, which reacts feebly.

On the R. side, the extensor communis digitorum, K S Z > A S Z.
On the L. „ „ „ „ .. K S Z = A S Z.
In all the other muscles of the forearms .. .. K S Z > A S Z.

Faradism.—Both forearms. Muscles of thenar and hypothenar eminences, the extensor ossis metacarpi pollicis and the flexor muscles react quite readily to moderate strength. The extensor communis digitorum does not react at all to the strongest current.

The supinator longus, extensor carpi radialis longior, and brevior, and the extensor carpi ulnaris react only to strong induced currents.

Mr. Gunn reports of the eyes:—" *Left side.*—The disc is practically normal, the upper edge a little indistinct. At the yellow spot the usual asterisk arrangement of small white spots characteristic of albuminuric retinitis. The *right* eye is normal.

"The right eye seems to have good vision, and there is no central colour scotoma. The left at first failed to detect green on central fixation, and first recognised it when it fell on the fundus a little outside the yellow spot; red was detected normally."

This patient presents superficially the aspect of a typical example of lead palsy. The history of the mode of attack, and the fact that patient has used no lead for six or seven years, and that he presents no blue line on his gums, afford practically conclusive evidence against this view of his case. And this is also confirmed to a considerable extent by the fact, that the supinator longus muscle shares with the extensors (although unequally) in the disorder.

The mode of attack, and the circumstance of his lower extremities being also affected at first, the feet not being able to be lifted but being dragged, point strongly to the probability, that he has suffered from multiple neuritis. The only apparent cause of this is the excessive indulgence in spirits which the patient freely admits. A suggestion from a speaker at the meeting of the Neurological Society at which I exhibited the patient, that it possibly might be an example of beri-beri, is negatived by the fact of the patient not having been on shore, and being the only invalid. It is not so easy to be sure that the patient is not suffering from the remains of an attack of anterior poliomyelitis. Against this view it will be noted that there appears to have been a good deal of numbness in the hands at first. The exact symmetry of the lesions again is much more in favour of polyneuritis, as is also the circumstance of the supinators being associated with the extensors of the wrists in the altered electrical reaction.

At the meeting in question, the President called attention to the huskiness of the patient's voice. Dr. Semon, who has since examined him at my request, reports as follows:—

"Both vocal cords move with respiration and phonation, but on phonation there is a curious relaxation of the left vocal cord, the inner border line of which is not straight but concave (paresis of the left internal thyro-arytenoid muscle). It is also seen, when the patient is told to produce a vowel (*a*) twice over in quick succession, that only the right cord moves energetically inwards and outwards, whilst the movements of the left are so lazy as to be almost imperceptible."

Dr. Semon is disposed to think that the laryngoscopic observation favours a diagnosis of multiple neuritis. The ophthalmic examination, although somewhat favouring the idea of chronic alcoholism, is not conclusive.

The case appears to me to be unusual, from the fact of the vastly more serious implication of the upper than the lower extremities. According to my experience, although the upper extremities are much more often than not involved, and frequently to a very serious extent, it is on the lower extremities that the severity of the attack in alcoholic neuritis appears to fall. So that in cases where the hands long continue useless it will ordinarily be found that the legs are still more powerless. In this instance the man's legs have apparently recovered perfectly, but his arms, though now improving, are quite incapacitated.

NOTE.—Since the above was in press I have seen in private practice a case of alcoholic neuritis, in which there still remain complete incapacity of one hand and partial disablement of the other, whilst the lower extremities have entirely recovered.

CASE OF ATROPHY OF THE FRONTAL AND PARIETAL LOBES OF THE BRAIN, CONSEQUENT ON LONG-CONTINUED PARALYSIS OF THE LIMBS FROM SPINAL DISEASE.

BY SEYMOUR J. SHARKEY, M.D., F.R.C.P.

S. P., æt. 37, was admitted into St. Thomas's Hospital under the care of Dr. Harley on the 2nd of August, 1884, and died there on the 3rd of February, 1887. She dated her illness from the preceding April, when she was confined of a stillborn child, since which time she had suffered from a pain in the back, and had wasted and lost power in her arms and legs, to such a degree that she could neither stand, nor carry anything in her hands. On admission, her legs and right hand were quite powerless, and she had retention of urine. On the 27th of March, 1886, a year before her death, pain and loss of power were noticed in her left arm, which gradually increased. She continued absolutely confined to bed during the time which she remained alive in St. Thomas's Hospital—a period of two years and seven months. During the whole of this time her legs were completely paralysed, and they became rigid and flexed, while her right hand was affected in the same way. Her left hand and arm were partially paralysed for the last year only. Sensation was retained throughout, except that on rare occasions some dulness was said to be noticed; but this was a transitory phenomenon. She had at first retention, and then incontinence of urine, with cystitis and attacks of high fever.

During the latter part of her life she put on flesh, and her general condition improved very much. Throughout, her mind was free from disturbance, and not in any way deficient. She died on the 3rd of February, 1887.

Post-mortem examination showed that the body was very well nourished.

The legs were rigidly flexed on the abdomen and at the knees: the fingers of the right hand were also flexed.

The lungs, heart, and liver were very atrophied, but otherwise presented no signs of disease.

The spleen was the seat of amyloid changes.

In the pelvis of the left kidney were several irregularly shaped calculi with centres of uric acid, and a deposit of phosphates externally. The pelvis of the kidney was inflamed, the ureter not. Amyloid changes were found in the straight vessels and Malpighian tufts. The right kidney and its pelvis were in a similar condition.

The bladder was inflamed, and its walls thickened.

Uterus and ovaries atrophied.

The brain[1] was healthy, so far as its structure, vascular supply, &c., were concerned; but there was very marked atrophy of the frontal and parietal lobes on both sides. The convolutions were thin, and separated from their neighbours by broad sulci, full of clear fluid. The atrophy was very striking, and was most marked in the upper extremities of the two ascending parietal convolutions of the right hemisphere, and in the superior parietal lobule. The same convolutions were most affected in the left hemisphere, but, in addition, the middle third of the parietal convolutions looked strikingly reduced in size. Microscopic examination showed that there was no disease in these convolutions, but that they were simply atrophied. The frontal convolutions on both sides were in a condition of atrophy similar to that seen in the parietal, but less in degree. In both hemispheres the occipital and temporo-sphenoidal lobes stood out in marked contrast, being well developed, and presenting an almost smooth expanse of broad convolutions, with very ill-marked sulci between them. The central ganglia appeared to be healthy, and those in the right hemisphere were examined in sections and found to be normal.

The bodies of five vertebræ—the sixth and seventh cervical, and the first three dorsal—were carious, and had partially disappeared. There were no collections of pus, but as the disease was most advanced on the right side, a marked curvature, with its concavity towards the right, had been produced. This had caused a displacement of the spinal cord to the left. The dura mater in this region was thickened, but it was not adherent to the spinal cord. The latter was softened for an area of about two inches, and below that level there was well-defined lateral sclerosis in the usual position. The nerves issuing from this part of the cord did not appear to be diseased, nor were the nerve trunks or large muscles of the limbs structurally altered, so far as one could judge from naked-eye examination; but the small muscles of the hand and some of the deep muscles on the flexor surface of the forearm were atrophied and pale.

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[1] See Plate, Fig 1.

The point of special interest in this case, for the sake of which I record it, is that it illustrates the separation in the cerebral cortex of the sensory from the psycho-motor centres. We have frequent opportunities of seeing this proved, either by the results of disease or of experiments on the cortex, lesions of which, in certain regions only, produce paralysis.

But it is a very rare event to get a good specimen illustrating this connection between certain parts of the brain and voluntary muscular action by the secondary effects on the cortex cerebri of experimental or pathological lesions of the cord, nerves, or muscles. The patient, whose brain I now show, may be said to have had voluntary muscular action reduced to a minimum for two and a half years. Her legs were quite paralysed, and incapable of being affected by voluntary stimuli during all that period; so was her right hand, and the inability to use the hand made it practically useless to move such muscles of the arm as were not paralysed. The left arm and hand were only affected for one year before death, and that partially. Being kept absolutely at rest in bed for two and a half years precluded the opportunity of bringing the muscles of the trunk into play, and even the head must have been subjected to but little movement.

The special senses, on the other hand, were all good and in use, and common sensation was little if at all affected. Hence if atrophy follows disuse, we should have expected those parts of the cortex which subserve voluntary muscular action to have wasted, while those which preside over sensation ought to have remained well preserved. Now without going into detail, the striking feature of this brain is the atrophy of the frontal and parietal, and the plump condition of the temporo-sphenoidal and occipital lobes. This, I think, is in accordance with our present views as regards the localisation of the sensory and motor centres.

We know too little of the physiology of the intellectual operations to say whether one part of the brain is more concerned in them than another. But even if it be held that the frontal lobes preside more especially over the processes referred to, one would not, on that account, have necessarily looked for mental deterioration in this case. For the absence of determinations to, and execution of, voluntary motor actions, might give rise to some atrophy of the parts concerned in them, without producing serious diminution of intellectual power.

A FATAL CASE OF TUMOUR OF THE LEFT AUDITORY NERVE.

BY SEYMOUR J. SHARKEY, M.D., F.R.C.P.

ON January 20th, 1887, R. G., æt. 41, came to my out-patient room at St. Thomas's Hospital, bringing with him a letter from one of the district Committees of the Charity Organisation Society, in which I was asked to say whether he should be sent to a Convalescent Home. He had already been in Bethnal Green Medical Mission Hospital for ten weeks, and after that at High Barnet Convalescent Home.

The patient informed me that up to the present illness he had been a healthy man; that he had never had syphilis.

His symptoms had first appeared during the preceding year (1886), and consisted of pains in the head, attacks of giddiness and "faintness," noise in the left ear, deafness, and some slight deterioration of sight. During the last five or six months he had lost a good deal of weight.

The headache had been in the vertex and frontal region. The giddiness was not only continuous, but increased paroxysmally, and in the paroxysms things appeared to revolve towards the left. These attacks occurred eight or nine times a day, and he was not free from giddiness even in bed. He had singing in the left ear and deafness, symptoms which had come on simultaneously.

In addition to the attacks of giddiness he suffered from what he termed "fainting fits." These attacks were ushered in by increased giddiness, but not by increase in the noise in the left ear; his sight got dim in them and he lost consciousness for some ten minutes. He never had vomiting, his appetite was good, and his bowels rather confined. His urine was free from albumen, and all his organs appeared to be healthy. On examination with the ophthalmoscope I found that he had double optic neuritis with great swelling of the discs. He had no paralytic phenomena.

Mr. Clutton, the Aural Surgeon to St. Thomas's, happened to be seeing patients in a neighbouring room, so I asked him to report on the man's condition.

His report was as follows: "Membrana tympani normal on both sides. Watch heard in right ear at two inches, in left not at all. Fork heard normally on right side, on left not at all, whether with auditory canal open or closed. Paroxysmal increase in the noise in left ear not synchronous with attacks of vertigo; this is against the disease being aural; but the vertigo is like that which is usually looked upon as aural, being both constant and increased in paroxysms. The examination with the fork would indicate that the disease is either in the brain, in the trunk of the auditory nerve, or in its peripheral expansion in the internal ear. For if it were in the middle or external ear, conduction with the fork would be intensified."

The presence of optic neuritis enabled me, however, to localise the disease within narrower limits, by eliminating the internal ear, and I came to the conclusion that the affection was intra-cranial, involving, if not necessarily originating in, the trunk of the left auditory nerve or the auditory centre in the temporo-sphenoidal lobe. A little more consideration at the time might have enabled me to localise it definitely in the trunk of the nerve; for if the centre only had been the seat of the disease, the patient would have had deafness, but not vertigo. The patient was subsequently admitted into the hospital, Mr. Nettleship kindly taking him into the Ophthalmic ward, as the medical wards were full. He was there treated with iodide of potash and mercurial inunction. His improvement was, however, slight.

He went out, and was again admitted on May 28th, 1887, into a bed kindly put at my disposal by Dr. Stone. He was then much in the same condition, except that he was thinner, and that his sight was worse. He had no paralysis and no anæsthesia, and his knee-jerks were brisk and equal. The left pupil was slightly smaller than the right, and there was thought to be slight deviation outwards of left eye when the patient looked straight forwards.

From time to time he had "fainting fits," in which he lost consciousness but had no convulsions. On June 17th, however, he had an attack in which some shaking and general rigidity were observed.

In August he had hallucinations of vision, seeing people round him and talking to them. Then for a short period he was violent, and refused food, and had to be fed through the nose. These symptoms, however, passed off. He occasionally complained of numbness in the gums, and his fits were more constantly ushered in by twitchings of the muscles of the limbs, especially on the right side. His sight too became so bad that he could scarcely see at all, and there was very slight paresis of the left side of the face. During the first three days of November he had fits in which he was unconscious as long as an hour, and in which his breathing became stertorous, and he died on November 4th.

At the post-mortem examination all the organs were found to be healthy, and the only pathological change present was an intra-cranial new growth, the nature and connections of which were as follows:

The tumour, which was rather larger than a horse-chestnut, occupied the angle between the cerebellum and pons on the left side, and in its growth it had formed a deep, cup-shaped cavity in the left lobe of the cerebellum. At first it was not clear where the tumour had taken its origin. It was found, however, to be entirely separate from the cerebellum, and only lying loose in a depression in it, which it had formed by pressure. It had pushed back the flocculus, and had flattened very considerably the left side of the pons and medulla oblongata.

It lay in the occipital fossa, and terminated in a short nozzle, which was lodged in a cavity in the petrous part of the temporal bone. This cavity showed no signs of disease, and the bone around it was quite healthy. It was, in fact, the internal auditory meatus expanded by the pressure of the tumour. The whole internal ear, together with the ossicles, was healthy.

After hardening the brain in spirit, the nerves were carefully traced. The fifth and the sixth were found a little displaced, owing to the alteration in shape of the pons and medulla. The facial was seen rising in its ordinary position, just below the tumour, and running along its anterior border towards the petrous bone. Over the lower surface of the growth posteriorly ran a nerve, subdivided into several bundles, and taking origin in the position usually occupied by the vagus. Close to the facial, and in front of the vagus, a nerve was found, originating in the ordinary position of the auditory: it entered the tumour, and formed the only connection which the latter had with any part of the surrounding nerve structures. Moreover, on section of the nose-shaped prominence which occupied the enlarged auditory meatus, the nerve was found, under the microscope, issuing from the mass of spindle-cells of which the tumour was mainly composed. Thus there can be no room for doubting, that the tumour grew in connection with the left auditory nerve.

The drawing by Mr. Shattock, which is here reproduced,[1]

V. marks the 5th Nerve.
VI. „ „ 6th „
VII.A „ „ Facial.
VII.B „ „ Auditory.
VIII. „ „ Vagus.

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[1] See Plate, Fig. 2.

gives a beautiful representation of the tumour, and its relations to the cranial nerves medulla oblongata, pons, and cerebellum.

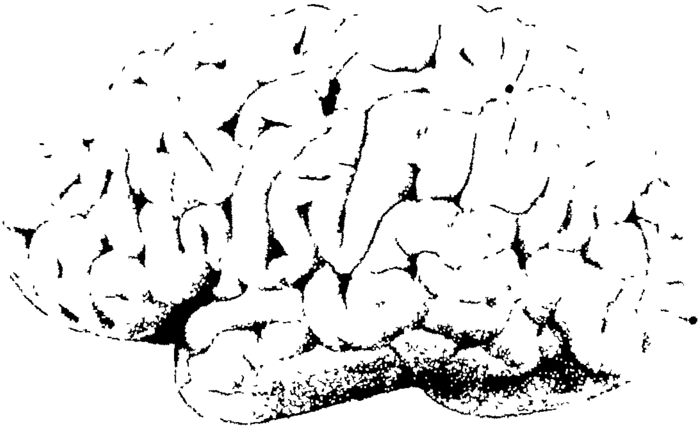
The interest of this case resides principally in the phenomena which ushered it in. The later symptoms were the secondary results of pressure on the neighbouring parts. Thus the shaking and rigidity of limbs which were observed in the epileptiform seizures which he had towards the end of the illness were due to the pressure of the tumour on the pons and medulla. The headache, hallucinations, and attacks of violence, and the optic neuritis with subsequent blindness, may occur in cerebral tumours occupying various positions.

But the very earliest symptoms of which he complained must be attributed to the disturbance of the function of that nerve alone on which the tumour was developed; and so we have a very interesting case of a pathological experiment performed on a nerve without any surgical operation, and consequently without the added disturbances which the latter may entail, and which often make the results of experiments inconclusive.

The earliest symptoms in this man's case were giddiness—continuous and paroxysmal—deafness and noise in the left ear, symptoms which are familiar to us as those of Menière's disease, in which the peripheral expansions of the auditory nerve in the labyrinth are affected. In experimental section of the auditory nerve in animals, and in destruction of the semicircular canals, the same intense giddiness has often been observed. But it has been attributed by some to the accidental lesions produced by the operation, and not to that lesion which the operators had in view. The present case, however, seems to me to be conclusive, as to the direct connection between section of the auditory nerve and the giddiness which follows it.

It has been noticed in the case of experiments that in time the giddiness and inco-ordination tend to diminish, and this was so in the case under consideration. The patient complained but little of it during the last few months of his life.

Another point of some interest is this, that the case would almost certainly have been put down as a case of Menière's disease at the time when I first saw him, unless an ophthalmoscopic examination had been made.



[illegible] Fig 1

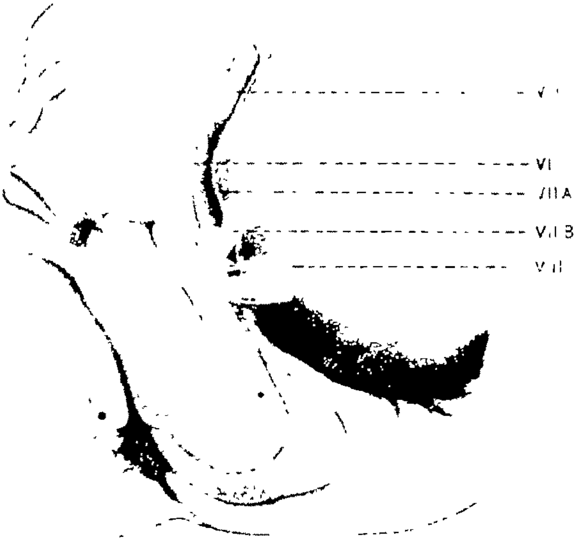


Fig 2

[illegible]　　　　Danielsson & Smith

EXPLANATION OF THE PLATE.

FIG. 1.—It is very hard to bring out in any single view, however well presented, the contrast between the Fronto-parietal and the Occipito-temporal regions which was so evident to any one who held the brain in his hands, and turned it in various directions. The plump condition of the occipito-temporal convolutions, and the well-filled spaces between them, presented appearances very different from the shrunken convolutions and the large furrows separating them, seen in the fronto-parietal region.

FIG. 2.—View of under-surface of Pons, Medulla Oblongata, Cerebellum, and Tumour.

V. represents the left 5th Nerve.
VI. " " 6th "
VIIA. " " Portio Dura.
VIIB. " " Portio Mollis

Entering the Tumour.

VIII. represents the left Pneumogastric coursing over the under-surface of the Tumour.

A CASE OF THROMBOSIS OF THE LONGITUDINAL SINUS, TOGETHER WITH THE ANTERIOR FRONTAL VEIN, CAUSING LOCALISED FOCI OF HÆMORRHAGE, WHICH PRODUCED REMARKABLY LOCALISED CORTICAL EPILEPSY.

BY VICTOR HORSLEY, B.S., F.R.S.

Surgeon to the National Hospital for Paralysed and Epileptic, &c.

The following case I publish, because it is valuable in localising the situation in man of Dr. Ferrier's area for the turning of the head and eyes to the opposite side, and at the same time the anterior limit of the upper limb area, together with the special representation of the segments of that limb at the anterior part of the region devoted to it.

Briefly summed up, the case is as follows: A man who had been suffering from tubercular disease of the bones for some months, suddenly developed symptoms of thrombosis of the longitudinal sinus, with cortical epilepsy as a result. The exhaustion produced by the epilepsy rapidly caused death. Unfortunately I never saw the patient during life, but I performed the post-mortem examination, for which I am very greatly indebted to the kindness of my friend, Dr. Lunn, and for the opportunity to publish the case in the interests of cerebral localisation, while I am also very much obliged to Dr. Courteen for the clinical notes.

J. J., aged 24, labourer, single, admitted into St. Marylebone Infirmary, Dec. 18, 1886. There was, on admission, chronic erysipelas of the scalp, a suppurating sinus situated over the right malar bone, and another over the left parietal bone just above and in front of the parietal eminence. These were freely laid open, but others developed in their immediate neighbourhood.

History.—The patient had been abroad some years, and a few weeks before admission was said to have knocked his head against a wall.

The patient, after admission, never seemed to recover, but gradually became weaker, in spite of extra diet, tonics, and the administration of iodide of potassium. Fresh suppuration was detected and treated vigorously, but without arresting the progress of the disease. A week before his death, the patient became much feebler and thinner, developed a hacking cough and became very drowsy.

March 4, 1887, 1.15 A.M.—The patient had a fit, followed soon afterwards by several others; each was of short duration, described by the Ward Sister as not lasting more than a minute. The fourth fit was observed by the Sister, who described it as follows: "The patient was lying on his back; first the head turned to the left, and he made a slight moaning noise; then the eyes turned upwards; he threw the left arm straight forwards, then upwards and outwards; the head then turned slightly to the right, and lastly both legs became convulsed, the mouth slightly open. Pulse 120."

The fifth fit occurred at 4.25 A.M.; but in this it was noted that in addition to the protrusion in extension of the upper limb, the left wrist was forcibly extended, or, according to the words of the Sister, "turned upwards," while the fingers were directed downwards. In fact, there was extension of the wrist with an interosseal position of the fingers. Several fits were observed after this last, but they gradually became weaker. The patient was quite unconscious from the onset of the first fit, and at 6.4 P.M. he died.

The movements observed, therefore, were first turning of the head to the left; then raising the arm at right angles to the trunk in complete extension, with extreme extension of the wrist and interosseal position of the fingers; gradual turning of the head to the right, and subsequently the rest of the body involved in the spasm.

Theoretically, we should have had to account for the particular primary movement and march of the spasm, in this case a largish lesion at the posterior border of the head and eyes area, where it becomes merged with the anterior border of the upper limb area in the right hemisphere, and then we might or might not have in addition a lesion in the left hemisphere at the highest point of the area of representation of the head and neck. According to the investigations of Dr. Beevor and myself,[1] these lesions, supposing both to exist, should have been situated in the following positions: the first, the larger, should have covered the posterior third of the middle frontal convolution, and should have to a slight extent invaded the anterior border of the ascending frontal convolution. The other smaller lesion should have been situated in the left marginal convolution in front of the vertical line carried up along the direction of the præcentral sulcus to the longitudinal sulcus. This point was also localised by Professor Schäfer and myself.[2]

As a matter of fact, both these imagined lesions were found to exist in the present case exactly in the spots posthumously diagnosed; but it is hardly necessary, perhaps, to point out that the second lesion was not, as it were, necessary for the production of the movement of turning of the head to the right towards the close of the fits, since it is well known that exhaustion of the head and eyes centre of the one side is followed by unresisted action of the centre of the opposite side, and consequently a corresponding change in position of the head. The accompanying diagram, which is outlined from a photograph of the brain very kindly taken for me by Dr. Wilson, late Senior Resident Medical Officer at the National Hospital for the Paralysed and Epileptic, Queen's Square, shows exactly the seat of the lesions.

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[1] 'Philosophical Transactions,' 1887 and 1888.'

[2] 'Proceedings of the Royal Society,' 1886. 'Philosophical Transactions,' 1887.

I will now proceed to describe the conditions observed in the autopsy.

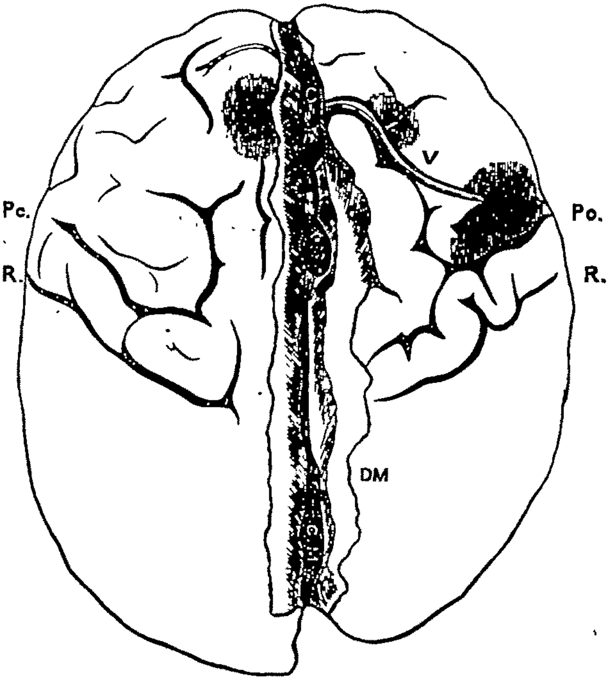
AUTOPSY.—Body well nourished. Rigor mortis not quite passed off. Over the right malar bone there was an old sinus leading to bare bone, and also a sinus over the left frontal bone, which similarly terminated in necrosed bone. There was a patch of caries, with destruction of the external table of the skull, midway between the posterior, superior and anterior angles of the left parietal bone; it formed an E-shaped area, the long side of which rested on the superior temporal ridge. The extremities of the area were each 2 centimetres in breadth. There was no trace of similar caries or necrosis in the rest of the skull.

On removing the calvarium, the surface of the dura mater appeared normal except over the anterior third of the longitudinal sinus, which appeared to be distended and discoloured. On the left hemisphere the dura mater was rather more prominent and bulging than on the right.

On slitting up the longitudinal sinus, there was found posteriorly a post-mortem blood-clot, partly decolorised, soft and dark black in colour for the most part, terminating anteriorly in a firm ante-mortem adherent clot, which extended from the junction of the middle and the posterior thirds of the superior frontal convolution, forwards as far as the crista galli. This clot was of a dark brownish-black colour, except opposite the middle of the superior frontal convolution, where it was very pale. The large vein on the right side which joins the sinus at this spot (referred to as the anterior frontal vein) was absolutely blocked by an ante-mortem clot, which as far as the superior frontal sulcus was of a black colour, but below that point of a pale greyish colour, and terminated below at the junction of the middle and lower thirds of the ascending frontal convolution. On the left side the veins entering the sinus from the external surface of the hemisphere were all pervious, excepting a small vein which joined the sinus opposite the junction of the anterior middle thirds of the superior frontal convolution, this small vein being blocked similarly to the large one on the right side.

This thrombosis of the sinus and veins caused the following lesions.

Right hemisphere.—The surface of the hemisphere appeared perfectly normal, except in the neighbourhood of the blocked frontal vein before described. The posterior sixth of the middle frontal convolution in its whole breadth was the seat of a hæmorrhagic extravasation of blood. The ascending frontal convolution was highly congested, especially in its anterior border; the membranes also of the superior frontal sulcus were congested along its posterior third, and there was a slight hæmorrhagic extravasation in the outer border of the middle third and the superior frontal convolution of this side (the right).



R.—Fissure of Rolando.
Pc.—Præcentral sulcus.
C.—Ante-mortem clot } lying in longitudinal sulcus.
C.M.—Post-mortem clot } lying in longitudinal sulcus.
D.M.—Edge of Dura Mater.
V.—Thrombosed vein.

The dark patches are the seats of hæmorrhage. The Sulci over the posterior part of the hemisphere are omitted for clearness.

Left hemisphere.—There was a dark black hæmorrhagic focus occupying the anterior half of the middle third of the superior frontal convolution for half its breadth. This, the only lesion in the left hemisphere, was situated (as before described) at the highest point of the area for the head and neck in the left hemisphere. It must here be mentioned that the whole of the surface of the left hemisphere was covered with a thin, tenacious, light red fibrinous exudation, which was adherent to the inner surface of the dura mater.

The rest of the central nervous system was perfectly normal. The other organs, moreover, presented no abnormality worth noting, except the lungs, which were both thickly studded with miliary tubercles. The occurrence of venous thrombosis in the cases of tubercular disease of the bones, &c., is too well known to pathologists to need special mention here, but we need the accumulation of many cases like the present to enable us to diagnose with certainty whether the cortical epilepsy is actually caused by hæmorrhage, as in the present instance, or, as would be quite as possible, by a tubercular nodule growing in the cortex, since in the latter case we could interfere with great benefit to the patient, whereas in the former case of course such treatment would be out of the question in the present state of our knowledge.

A CASE OF CHRONIC MENINGITIS, PROBABLY SYPHILITIC, AND CAUSING PROGRESSIVE DEMENTIA.

BY FRANCIS WARNER, M.D., F.R.C.P.,

AND

FLETCHER BEACH, M.B., M.R.C.P.

WILLIS C., a boy aged 7 years, first came under observation as an out-patient at the East London Hospital for Children in January 1879.

He was considered healthy at birth, and was suckled eleven months; as an infant he suffered from snuffles, thrush and sores about the nates. He never had fits. He had been attacked with measles and whooping-cough, but got well over them, and appears to have been a strong boy. He went to school, did his lessons fairly well, and could write; he continued bright and well till about eight or nine months before he was first seen. About April 1878 his mother began to notice a certain difficulty in his movements, which continued. At the same time he began to complain of headaches, and to cry for slight causes; a sudden noise would disturb him, and the sight of certain objects or animals, or even of other children, would frighten him. He still continued at school, but the teacher said he had gone back in his learning.

When first seen he could not protrude his tongue properly; his teeth were ground, the upper incisors lost, and it was ascertained that he had the habit of tooth-grinding, especially at night. He often complained of being tired, and of having headaches; sometimes he vomited. He was not mischievous at this time. His head was well-shaped, 20¼ inches in circumference; his weight was just 3 stone. His heart and other organs appeared healthy.

To summarise the facts observed during the next six months. When sitting on the floor he had great difficulty in getting up, and could not do so without using his hands; he could go upstairs well, but had great difficulty in coming down. The calves each measured 8½ inches in circumference; the knee-jerk was well marked on either side; his gait was very stiff. He would sometimes grind his teeth during the day as well as night.

Purgatives occasionally brought away a few round worms, but their expulsion had no material effect upon his condition. Mercury with iodide of potassium was given almost continuously through the year.

In February 1879, it was noted that at times he seemed in a dormant condition, as if mentally lost; the left pupil was usually larger than the right, but they varied in size.

In March he was reported to have had "an attack," the first that had been noticed; he trembled, and would have fallen had he not been supported; he did not lose consciousness. When seen he was stupid, and would not shake hands. Soon after this he became weaker, dull and stupid. He could speak, but had some difficulty in swallowing; he would fill his mouth without swallowing the food.

In June he vomited occasionally; he walked badly. There was distinct mental deterioration, he could hardly say the alphabet; occasionally the bowels acted involuntarily; he had grown stouter.

In July 1879 he was admitted an in-patient under Dr. Eustace Smith, to whom we are indebted for the following notes.

"A well-nourished boy, with healthy complexion. He is very troublesome, often trying to get out of bed, often shrieking, apparently neither from pain nor from passion. He talks only nonsense, neither asking for food nor for anything else. When given toys he is amused with them for a few minutes, and then ceases playing with them, but does not break them or throw them away. He can run down the ward, but does so as if frightened. The facial muscles all act well, there is no paralysis of any ocular muscle, the left pupil is larger than the right. Sight appears good, no signs of keratitis. On tickling the soles of the feet, reflex action is well marked. He is generally good-natured, but does not appear to know what he is doing: more he likes music. He often vomits and is very constipated.

"When given a picture-book, he will play with it, but keeps it upside down. At times, however, he attempts to hurt other children, and will knock and bruise himself." He was discharged as being unmanageable.

During the remainder of this year he was frequently seen; at times he was very irritable and unmanageable, and his hands became very cold and blue. Sometimes there was vomiting, but no complaint of pain. The signs of intelligence varied; in December he was taken very ill, vomiting the whole of one day, after which he lay in a condition of torpor, and for the two succeeding days was laughing, singing, and grinding his teeth.

January 19*th*, 1880.—He was brought to the hospital much worse. He came into the consulting-room looking dull and silly, grinding his teeth, his face and hands bluish. He could stand and walk a little; he walked to the hospital with difficulty. He was dirty in his habits, and had to be fed. He took no notice of questions put to him, and would not look at any one speaking to him, nor protrude his tongue when asked to. He could drink well. Pupils were large; the heart was very feeble. No fits. He was again admitted in-patient under Dr. Eustace Smith. Another attack occurred in the hospital, in which he was unconscious for six hours; no convulsions; at times he would scream and laugh. He was discharged as a hopeless imbecile.

In June 1880 he was admitted in-patient at the London Hospital. He was still well nourished, inclined to be noisy, said nothing but "Mamma" and "Dadda." He was constantly grinding his teeth, and never would play. He could stand, and there was no palsy of any cranial nerve; he could apparently see and hear. His eyes were frequently examined ophthalmoscopically, but no changes were seen.

In September 1880 he was admitted into Darenth Asylum, and the following note was taken: "A thin, dark-complexioned boy, aged nine years; manner peevish, with dirty habits. He can hold objects with his right hand, but not with his left, which usually has the thumb turned in; the second toe of his left foot is drawn down. He cannot stand without support, and his gait, when supported, is jerky. Pupils are sluggish, and speech has been lost. No disease of spine."

He became very weak and emaciated, and died January 19th, 1882.

The following history of the family was obtained: His maternal grandmother was epileptic. His mother, aged 43 when the patient was first seen, had been liable to spectral illusions, generally seen at night when going to sleep; she had been liable to headaches.

The father, aged 44, a sailor, had been under the care of Dr. Stephen Mackenzie, who informed us that he found signs of syphilis in the man from a sore contracted in 1851; he had a temporary attack of hemiplegia when 13 years old. There had been six offspring in the family: (1) miscarriage; (2) ditto; (3) born at eight months, then died; (4) the patient; (5) a girl, healthy; (6) a miscarriage.

Autopsy (twenty-seven hours after death).—Body much emaciated; bed-sores. On removing the skull-cap, the dura mater was seen, and this was found to be adherent to, but easily separable from, a subjacent false membrane, the surface of which was glistening, and of a pink colour. This false membrane was about two lines thick on the right side; on the left side it was thicker. It had evidently been formed some time, but the upper surface appeared to have been recent. It was attached here and there to the upper surface of the pia mater by thin membrane, but was easily separated everywhere from the brain. There was some fluid beneath the membrane. After the brain was removed, the membrane could be traced along the floor of the skull as far as the superior margin of the petrous bone, but it did not line the posterior fossæ. In front, on the right side of the floor of the skull, it was thin, but at its posterior part it averaged from 6 mm. to a quarter of an inch in thickness; on the left side it was not so thick. The upper surface of the brain was congested, and the vessels of the pia mater were injected.

The brain weighed 1 lb. 11 oz. Convolutions were not so simple in character as in cases of congenital imbecility.

The other organs were normal, except the left lung, which was collapsed from old pleurisy.

The wood-cuts of the brain are from photographs taken for me by my friend, Mr. W. T. F. M. Ingall, and represent the brain after being in spirit, and then in a weak solution of chloride of zinc for some time. They are two-thirds of its actual present size.

This case is interesting in many respects. It was clearly one of acquired imbecility, due to chronic meningitis, probably syphilitic in its origin, though prolonged mercurial treatment appeared incapable of arresting the disease.

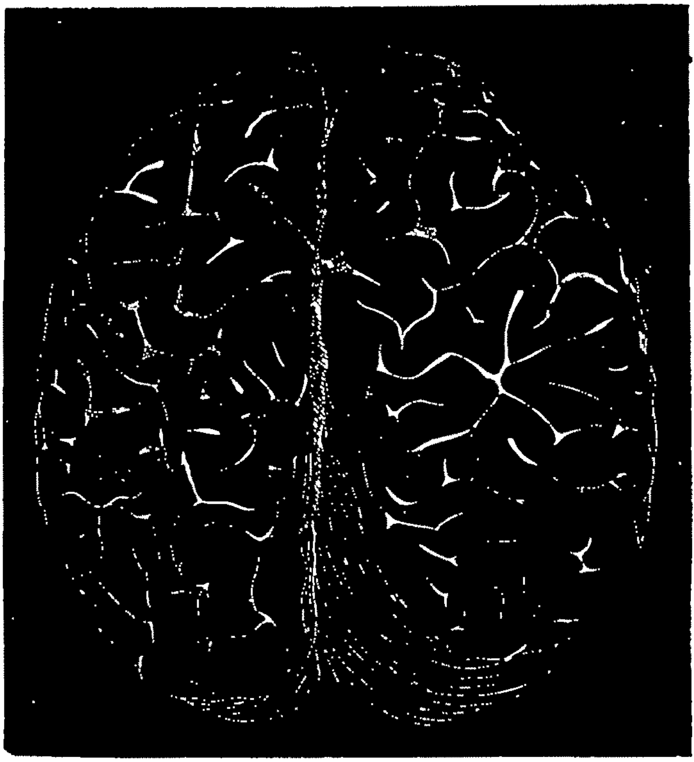


FIG. 1.

The case is representative of many in Darenth Asylum, where, owing to the influence of heredity, indicated here by the history on the maternal side, the child is born with an unstable brain, easily upset by any cause. In this case the cause was meningitis, probably due to syphilis inherited from the father. Syphilis is not a common exciting cause of imbecility, but according to Heubner, hereditary predisposition to nerve-disease appears to exert an influence in determining the syphilitic poison towards the nervous system. As is usual in such cases, the brain is of comparatively small size. The determining cause of the onset of the pachymeningitis does not appear clear from the history.

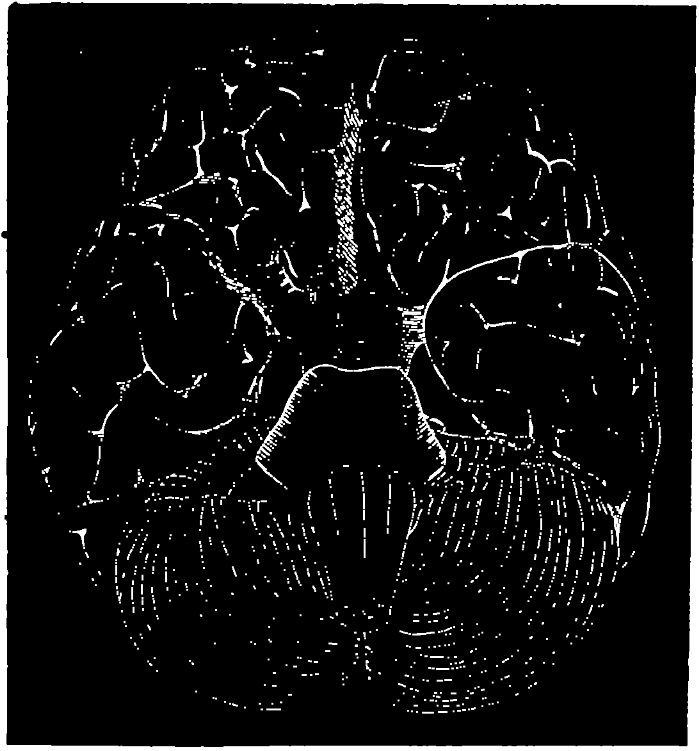


FIG. 2.

The following references are given, illustrative of the connection between syphilis and idiocy and imbecility:

Ireland: 'Idiocy and Imbecility.'
Griesinger: 'On Mental Diseases.'
Lancereaux: 'Traité de la Syphilis.'
Hughlings-Jackson: 'London Hospital Reports,' vol. i., 1864.
Maudsley: 'Pathology of Mind.'
Bucknill and Tuke: 'Manual of Psychological Medicine.'
Mendel: "Ueber Hereditäre Syphilis in ihrer Einwirkung auf Entwickelung von Geisteskrankheiten."—'Archiv für Psychiatrie,' Bd. I., 1878–79.
Bury: "The Influence of Hereditary Syphilis in the Production of Idiocy or Dementia."—'Brain,' Vol. VI., 1883.

A CASE OF ALMOST COMPLETE ANÆSTHESIA WITH ATAXIA OF THE LIMBS.

BY C. E. BEEVOR, M.D., M.R.C.P.

Assistant Physician to the National Hospital for the Paralysed and Epileptic, and the Great Northern Hospital.

AMY S., aged 24, noticed seven years ago that she gradually became weak in the right hand and arm, and at the same time they felt numb; she frequently dropped things, and had difficulty in picking up small objects. Two months later she noticed that the right leg felt heavy and numb, and she had some difficulty in walking, and it dragged after her. One year after the onset she found out that she had lost feeling in the whole of the right half of the body. Two years from the onset the left arm began to be affected with numbness and weakness similarly to the right arm; the left leg was subsequently affected, though to a less degree than the right.

She had measles when 15 years old; she never quite recovered her health after this illness, and her present illness came on about two years later.

At different times she has had sharp shooting-pains in the right arm, lasting only for a second or two.

Since onset she has had some difficulty in holding her water.

She first came under my observation in June 1884, and she has since been an in-patient in the National Hospital for the Paralysed and Epileptic, under Dr. Hughlings-Jackson and Dr. Ferrier. At that time—three years ago—she presented the following condition:

She was a well-made girl, and though rather apathetic, she did not present any characters pointing to hysteria.

Motor system.—Face normal, no paralysis of the motor cranial nerves. Pupils equal, reacting to light and accommodation.

Right upper limb is somewhat wasted, and especially the extensors of the wrist, the deltoid and triceps muscles. She has much difficulty in separating the fingers and in extending them; the grasp is weak, but flexion of the fingers is much better than extension; she has great difficulty in picking up small objects with the right hand. Flexion of the elbow is better than extension; she can abduct the shoulder to the horizontal line, but not beyond this. She has most of the movements of the limb, but they are performed very slowly, and with much hesitation.

Left upper limb is less wasted than the right, but there is not much difference in the measurements of the forearms and the middle of the humerus of either side. The grasp is better than on the right, and all the movements are better performed, but they are slower and more hesitating than normal. She can, however, pick up a pin with the left hand.

The *Lower limbs* are well developed, and she has all the movements in them, but the left is stronger than the right leg. She is able to walk a mile with assistance.

Knee-jerks are very excessive; *ankle-clonus* very well marked on both sides. *Plantar reflexes* are normal.

Sensation is very much affected nearly over the whole body, but especially on the right side.

The right arm is anæsthetic to tactile sensation throughout, and is analgesic to painful sensations as far as the elbow; the loss of tactile sensation extends up the neck to the face—where at times she can localise correctly, but often not—and above to the middle line of the head, and behind to the spine; the trunk is anæsthetic on the right side to the middle line.

The right leg is completely anæsthetic to the knee, and to a less degree in the thigh.

The left arm is anæsthetic to the elbow, and analgesic to the wrist; the left leg is only slightly anæsthetic to the knee, while in the left trunk she can localise tactile sensations fairly well.

Ataxia and *loss of muscular sense* are very marked. She cannot stand with the eyes closed, but staggers at once, and falls.

When the eyes are closed she cannot touch the nose with either hand, but she has more difficulty with the right than with the left upper limb.

She has no idea, with the eyes closed, when the right hand or elbow are moved passively, or when the left fingers and wrist are flexed, but she can just tell when the right shoulder and the left elbow and wrist are similarly treated; she cannot tell the position in which the right forearm or the left hand is placed. With the eyes shut she cannot appreciate different weights put into the hands.

In the legs she cannot appreciate movements, or the resulting position when the right foot and knee are bent, or when the left foot is moved.

The muscles of both forearms react normally to the faradic current.

Sight normal; no colour-blindness; field of vision not affected. Optic discs normal.

The above account refers to her condition in 1884.

Present condition has not altered much, but the anæsthesia is more marked than previously.

Right hand is rather more wasted, and has a tendency to become "clawed," and she is unable to separate the fingers; it takes her some time to extend and flex the fingers, as if she had to overcome some obstacle.

Sensation to touch and pain is almost completely absent over the whole of the right side.

The left arm is anæsthetic to the middle of humerus.

The left leg is completely anæsthetic, but she can feel painful impressions. In lying down she can only feel the left half of the bed, and it seems as if she were lying on the edge of the bed, and would fall out towards the right side. When sitting down she cannot sometimes feel the chair at all, and with the eyes closed she seems to be floating in the air.

Electric stimuli are very imperfectly felt on the right side of the body.

She has the same degree of power whether the eyes are open or closed, and she can kick out with either leg equally well with eyes open or shut.

Plantar reflex is now absent on both sides.

The *knee-jerks* are excessive, and *ankle-clonus* is present on both sides.

In the above case the chief points are :—Almost complete anæsthesia of the right half of the body and limbs, and a considerable amount of anæsthesia of the limbs of the left side and to a less degree of the left half of the body; loss of muscular sense as regards position and the appreciation of weights, together with marked ataxia in all four limbs, when the eyes are closed. Associated with these symptoms there is very little paralysis.

She is able to walk a mile leaning on another person's arm, and although she has lost the finer movements of the right hand, and she has some difficulty in moving the other joints of the right arm, there is no marked paralysis.

There is ankle-clonus and excessive knee-jerks on both sides, but there is no rigidity of the lower limbs.

The case is somewhat similar to those referred to by Dr. Bastian in his paper on "The Muscular Sense" read before the Neurological Society last December, and especially the case of Remigius Lens, published by Spaeth.[1]

The diagnosis of the case is difficult, the symptoms are rather like those of hysteria; but she has been in the hospital twice as an in-patient, and all treatment has failed to produce any improvement.

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[1] Beitr. zur Lehre von der Tabes Dorsalis;" Tubingen, 1864.

TWO CASES OF INSANITY DEPENDING ON SYPHILITIC DISEASE OF THE ARTERIES.

BY GEO. H. SAVAGE, M.D.

I HAVE ventured to record the following cases as very good examples of two very different classes of cases which are met with in asylums; in both there was no doubt about the history of syphilis, and there is no doubt about the changes which were found after death being such as have been usually attributed to that disease.

In the first case, progressive dementia associated with local softenings in the brain was present; while in the second, the disease of the arteries was followed by the special form of degeneration which we call general paralysis of the insane.

Why in one case the process should be one of simple decay, while in the other it was associated with a period of excitement, and why with this progressive disease there was a temporary lull, I am unable to explain.

No form of treatment is of the least use when arteries have become as diseased as they were in both these cases, and the history of syphilis in no way made the prognosis better.

In both cases there was a distinct history of nervous instability in the families.

Syphilitic Dementia with localised Softening, with marked Disease of Arteries.

Notes before admission.—M. W. Single. Worked for living as a clerk. He belongs to a noted family which has produced distinguished men as well as spendthrifts. A cousin married a notorious woman, who had a child, which child deprived this man of the inheritance. He was an odd boy at school, where it is said he drank. He contracted syphilis at the age of 17; had no serious secondary symptoms. He led a solitary life, and drank very heavily till about four years ago (1883), when he reformed. He had a fit about two years ago (1885), which left him with right-side hemiplegia and aphasia. He regained power in his leg and arm, but his speech has been defective ever since. He was in no way more peculiar than usual till November 1887, when he began to fancy things which were not true. He would lie in bed by day, and at night he would wander round the square where he lived. He forgot his appointments and neglected his duties. Dr. Savage saw him Dec. 16th, 1887, and was told there was some suspicion he was malingering because some women had accused him of having relations with a child below the legal age. He was said to have been given of late to the lowest female company. He fancied he was in debt, and had hallucinations of smell.

On December 17th he was admitted to the hospital.

On admission.—Paternal first cousin was insane. Father died of asthma and thrombosis.

Patient was eccentric, a good linguist and well educated; always melancholy, and avoided outdoor exercise. Drank hard from 1875 to 1879. Paralysis two years ago supposed to be due to drink. Right arm lost power. Speech affected. Had syphilis when about 17 (now 37). Has been abstainer for four years.

Time of earliest symptoms about three weeks before admission. He was silent and melancholy, and said he was in debt, which was not true. He wandered about, became more silent, refused food, saying it was poisoned and that his teeth were all gone. He would not wash, and said he had attempted his life. He said his coffee and food were unpleasant.

Tongue clean, pale; no tremor. Appetite bad. Wet and dirty. Urine 1020, acid, phosphates, no albumen. Pupils equal dilated, reacting to light.

After admission.—Tall; dark brown hair and beard; grey eyes; face sallow; tongue clean, no tremor; depressed anxious expression. Refused to answer any question; was restive and inclined to be violent while being questioned. Restless during examination of lungs, but would not speak.

Dec. 19, 1887.—Sits unoccupied. Takes food badly. Wet and dirty. Says he cannot talk. Sleeps badly.

Dec. 23*rd.*—Fell down in the gallery this morning. He was apparently conscious, and had not had a fit, but looked faint and ill. He was found to have lost power, and there was anæsthesia of the left hand. He formerly had right hemiplegia and aphasia.

Dec. 24th, at 6 P.M.—Pulse feeble; loss of power in left arm; eyes turned to the right. Anæsthesia of left side, and marked coldness compared with the other. Breathing stertorous; fed with nose tube.

Dec. 25*th.*—No change, except that the right hand is kept clenched, and there is ankle-clonus of the left ankle.

Dec. 26*th.*—Pulse feeble; tongue and lips dry and foul; eyes directed to right. Right hand clenched. Anæsthesia of the whole of left side. Temperature of both sides equal, 99°. Fed with nose tube. Pot. iod. gr. x, liq. hyd. perchlor. ♏ 60, t. d. s.

Jan. 2nd, 1888.—Some improvement; more colour in face; breathing normal; eyes still turned to right, but takes hold of a proffered hand with his right, which is less firmly clenched, and can be opened voluntarily. Some ecchymosis in the outer canthus of the left eye. Mouth very dry; right half of tongue thickly furred; left half clean. Distinct pallor of left optic disc.

Jan. 4th—Not so well; breathing again stertorous; pulse small and rapid; bowels acting frequently; motions very offensive. 11 P.M.—Was found cold and almost pulseless, but rallied gradually.

Jan. 5th.—Evidently dying. Pulse feeble; surface cold; bowels acting constantly. Died at 9.30 P.M. Shortly before death he had some clonic spasms of the left side.

POST-MORTEM.—*Sixty-two hours after death. Weather mild.*

Body fairly nourished; rigor mortis in legs; no bed-sores or marks of injury. Brain, 46 oz.; heart, 14 oz.; right lung, 40 oz.; left lung, 30 oz.; liver, 59 oz.; kidneys, R. 6 oz. and 5 oz.; spleen, 6 oz.

Calvaria thick; dura mater apparently normal; slight excess of sub-arachnoid fluid. P. M. peels readily; no sign of gummatous infiltration at base. In left internal carotid is a long, recent, loose clot.

In right middle cerebral artery, about an inch from its origin, is the characteristic appearance of syphilitic arteritis, the vessel (or rather two parallel vessels) being impervious at this point. The vessels of the opposite side appear to be normal. No atheroma seen. On making a section of the brain the puncta cruenta are very marked, and just outside the frontal end of the caudate nucleus, and before reaching the level of the ventricles, there is seen to be on the right side a spotted patch. On cutting down there is found to be a small cavity, the size of a bean, situated at this spot, and containing some grumous fluid. The walls appear to be smooth, as if the cavity were not quite recent. The surrounding white matter is somewhat soft and pulpy. On the left side there is seen to be an old cavity with smooth walls, apparently the result of a former softening. This occupies the internal capsule just outside the caudate nucleus at the level of the anterior end of the optic thalamus. There is no sign of any hæmorrhagic condition, and no blood pigment about. Rest of brain apparently normal.

Both lungs congested and œdematous; right hypostatic consolidation at base; heart pale and flabby; valves normal; kidneys large and pale; spleen and liver normal.

The above case may serve as a clinical contribution to the localisation of function, and also as a typical case of mental breakdown associated with syphilitic disease of the arteries. The patient belonged to a highly nervous family. He contracted syphilis when about seventeen, and did not suffer from the disease of the arteries for eighteen or twenty years. One of the earlier of the marked symptoms of degeneration was exhibited by excessive sexual desire, or loss of control of this desire. He fell into very low company, and was threatened with trouble, in consequence of frequenting the society of very young girls.

On first seeing the patient, it was difficult to decide whether he was malingering to escape from justice, and but for the traces of the aphasia, with the history of the fit, I should have been in doubt. As has been recorded, he had a second fit, which proved to be the beginning of the end.

There were two local softenings, one which accounted for the right hemiplegia and the aphasia; the other, of more recent origin, was in front of the corpus striatum—in fact, in the area which Dr. Hale White has pointed out as possibly being associated with the control of the body temperature. In this case there was, after the loss of power on the left side, a marked change in the temperature on the left side; but, beside this, the left half of tongue was cleaner and moister than the corresponding right half. There was pallor, too, of the left disc.

The innumerable local wastings which may result from syphilitic arteritis make the careful recording of such cases of service toward the elucidation of the specific function and relationships of the parts of the brain which depend on local blood supply, so that such cases may enable us to separate distinct areas of blood supply, and compare them, with special areas of functional activity or irritability.

Geo. S. R., admitted, Oct. 16th 1886.

Maternal grandmother epileptic. Mother poisoned herself at time of climacteric, had previously suffered from metrorrhagia, followed by anæmia. One paternal aunt died of phthisis.

Patient's History.—Unusually brilliant. Athletic in Cambridge eleven. Sober. Had syphilis while at College. Slight secondaries. Slight sunstroke some years ago. Disappointment at not gaining an appointment in July.

Earliest symptoms, end of July last.—Sleeplessness following shock. Given large doses of chloral and opium, for about a month, during which time he had two or three attacks of coma lasting about two days (thirty hours). Loss of memory and of nerve power; excitement and violence at times.

On examination;—Tongue flabby, tremulous, slightly furred; appetite good; bowels regular; lungs normal; heart normal; reproductive organs normal. Eyes good, pupils large, react to light; right larger than left. Ears and hearing good.

Common sensibility good; says he can't feel; temperature normal; reaction to questions slow or absent; depression; memory, recent and remote, confused. Sleep disturbed, dreams not marked. Walk wanting in firmness and elasticity, pigeon-toed; reflexes very brisk; knee-jerks.

Dynamometer, R. 65, L. 53.

A tall, somewhat slim, fairly well-nourished man. His hair, beard, moustache and whiskers are black and coarse, with here and there a grey hair. His complexion is fresh and ruddy; his eyes dark brown, with widely dilated pupils, the right pupil being slightly the larger. His aspect is somewhat vacant. He goes about with half-open mouth and elevated eyebrows. His tongue and lips are tremulous, his speech explosive and blurred. Both hands are somewhat blue and cold. When turning suddenly, he staggers; his walk is feeble and pigeon-toed; reflexes brisk. Seems very confused; cannot say how long he has been here, how he came, or where he is. Is quiet; occasionally plays chess, but as a rule is unoccupied. Takes his food well, but is a little restless at night. Has a tendency to collect rubbish. Uses the spittoons to pass water into. Does not show any tendency to suicide or violence. His memory for recent and remote events is confused; he does not answer questions readily. One cannot get any evidence of hallucinations, but he has a delusion that he is watched by a policeman. The patient, in short, appears to be suffering from general paralysis of the insane.

Oct. 17*th.*—Believes that the night watchmen are policemen who were sent to watch him. Still in a quiet stupid condition.

Oct. 20*th.*—No change; moved from No. 1A to No. 3 Infirmary.

Oct. 27*th.*—A little brighter; answers questions better. Still unoccupied and quiet. Recognises his wife. Circulation better.

Oct. 31*st.*—Quiet and unoccupied, but looks much brighter; less taciturn.

Nov. 9*th.*—Memory bad; thinks he has been in Bethlem six weeks. Quiet and rather apathetic; reflexes brisk; right pupil larger than left.

Dec. 8*th.*—Leave of absence three weeks. No marked mental improvement since last note.

Dec. 29*th.*—Extension of leave, three weeks.

Jan. 3*rd*, 1887.—From his father's letter of this date it appears he still entertains the delusion of his wife's unfaithfulness, and he hears voices, otherwise he appears in good health.

Jan. 19*th.*—Extension of leave three weeks.

Feb. 9*th.* Ditto. Ditto.

March 2*nd.*—Letter from patient's father announcing his complete recovery. Discharged well.

July 17*th.*—*Re-admitted.*—After leaving as above he kept well till May, when he was in Greece. He had intermittent fever a month before, and suffered from headache. In May he left his wife in Corfu, and went to Athens. On the way he was excited. He was away from his friends a week. On the previous Sunday he had made many mistakes in reading the lessons, but preached a very good sermon. He said he was worth millions, that he was captain of the vessel, and Christ. He recognised his wife when brought home, but was very violent. Was always attacking his attendants. He had a great contempt for the Greeks, and used to fight them on every opportunity. They were all very frightened of him. He used to say that an Englishman should be equal to twenty Greeks, and he tried to demonstrate this on every occasion.

He said he would ask God to hang him. He never showed any tendency to suicide. He attacked every one who came near him most violently. Always liked to be with his wife.

Before the last attack he thought a book was lying on the table, when it was really at the end of the room.

Had prickly feeling in arm and leg. Had difficulty in opening his jaw when first beginning to eat. Tongue furred, flabby. Lungs and heart normal. Eyes normal, pupils equal. Reaction to questions; not to the point; voluble, profane. Maniacal. Restless. Walk natural.

On admission.—Tall, dark, powerfully made man. Black eyebrows and beard. Very violent at times, constantly talking. His talk consists mostly of obscenities, with biblical quotations. He seized Mr. J. round the neck, and said he would hug him, and proceeded to illustrate his words. He collects scraps of paper, leaves, sticks, and stones, and fills his mouth with them as well. If interfered with, he spits at every one within reach. He occasionally adopts an ecclesiastical tone of voice, and speaks of damnable heresies, love of the girls, mixing in Greek and Italian words.

He says he has great wealth. Whilst coming from Corfu he wanted to get out, to show that he could walk on the sea.

July 19th.—Still violent, destructive and mischievous.

July 21st.—Says this is the most excellently managed hotel he was ever in. He has gloves on, and requires an attendant to constantly watch him.

July 23rd.—Very violent. Says a voice tells him to die. "If you wait a minute you will hear the voice. There it is! a million pounds for this man!"

July 28th.—Violent.

August 2nd.—Tore up his bed last night. Talking constantly to himself and swearing. In side-arm dress.

Aug. 5th.—Up in airing court; quieter.

Aug. 6th.—Much quieter; says he is going to India.

Aug. 8th.—Says he is the "Bishop of Chester and Hawarden." "Many thanks for getting me the appointment. It is worth a million a year. You know I did not seduce that girl, I married her, the daughter of the Governor-General of Cyprus. It was all a mistake, you know I would not do such a thing." 3.15 P.M. Hyoscyamine, gr. $\frac{1}{80}$. Was very quiet while it was given, and afterwards.

Aug. 9th.—Says he felt much better after the injection. "Slept for two years and a half."

Aug. 12th.—Very noisy when spoken to. Began to cry, and says he never seduced that girl.

Aug. 15th.—Hyoscyamine, gr. $\frac{1}{40}$. Was restless after it, and lavish in his curses to the —— —— who gave it him.

Aug. 16th.—Anxious not to have another injection, it made him feel so ill.

Aug. 19th.—Has been tearing up and chewing his bedding. When asked why he did so, he said, "It is locusts and wild honey. A fellow who does not eat locusts and wild honey when he can get it, must be a d— fool."

Aug. 22nd.—Vomiting several times this morning. Began before breakfast to go to bed. Much bile vomited.

Aug. 23rd.—Not sick to-day. Much quieter. Conjunctivæ rather yellow.

Aug. 24th.—He vomited once to-day after dinner, but seemed better later in the day.

Sept. 1st.—This morning he was walking about in the airing court, talking much as usual. Said he felt quite well, and made some of his usual statements about having "married the girl." He looked much thinner than when he came in, but otherwise seemed fairly well. At 4.30 P.M. he was sitting in the gallery with a newspaper before him. About 5 o'clock he was noticed to slip off the seat he was sitting on; he became unconscious; no cry. Was then put on the bed; his left arm became drawn up, and his left leg straight, but twitching. The right arm and leg were flaccid; face not affected. Breathing stertorous, temp. 98·4. Later breathing became very irregular and stertorous. He had several attacks of congestion of face, with blueness of lips and tongue and stoppage of breathing, These attacks ran into one another without definite convulsions. He was unable to swallow, and mucus collected in the back of his pharynx and seemed to choke him. It improved the breathing when he was turned on his side and the mucus wiped out of his mouth. About 7 o'clock his left arm was flaccid, and it was then found his left leg was cold, and right hot. Temp., R. axilla, 103. Injectn. Æther-hypoderm. ♏ 30 at 9.5. At 9.30 the eyes were examined, and the only thing noticed was congestion of retinal veins.

He remained in an unconscious state, with occasional stoppage of pulse and breathing, the breathing being slow, 10 to a minute, and stertorous. He died at 11.35 P.M.

POST-MORTEM.—24 *hours after death, weather cool.*

Rigor mortis in legs, not in arms. P. M. lividity on back. No bed-sores or bruises. Encephalon, 50 oz.; heart, 12 oz.; liver, 7 oz. Lungs, R. 27 oz., L. 26 oz. Kidneys, R. 5½ oz., L. 6 oz. Spleen, 4 oz.

Calvaria irregular in thickness. In region of middle meningeal arteries on both sides the channels for the vessels, especially the small ones, are very deep, and in some instances tunnels. Attachments of superior longitudinal sinus very prominent. Dura mater somewhat adherent to bone over both parietal eminences and surface of frontal region. Arachnoid rather thick and opaque; no excess of fluid. Pia mater somewhat thick, but peels fairly easily, except over each superior frontal convolution, where there is some adhesion. Some congestion of veins at occipital region. No marked wasting of convolutions on surface. Left vertebral artery very much smaller than right. At commencement of basilar artery is a round projection of the vessel about the size of a small pea. On section, this is found to be due to the thickening of the coats of the vessel, and at this spot the vessel is blocked by an old clot which has a cavity in the centre. Rest of basilar artery occupied by a recent soft black dot. No abnormality observed in other cerebral arteries. The ventricles of brain not distended, and interior normal to naked eye, except that the grey matter, especially in the motor areas, is extremely pale and almost white in places. This was most especially so in the left ascending frontal and parietal convolutions. Thorax: lungs distended, normal, except for some œdema at bases. Heart flabby, much post-mortem staining. Slight atheroma of aorta. No valvular disease. Very little clot.

Abdomen.—Liver normal; kidneys normal; spleen normal; intestines healthy; vermiform appendix situated behind cœcum, running downwards behind peritoneum on psoas muscle. Stomach, some p. m. staining and injection of vessels. No ulceration. No sign of gastritis or ulcer.

The above is a case in which general paralysis of the insane occurred in a man who has had syphilis in his early youth, and who had no trouble from secondary symptoms. In his case there was insane inheritance.

The earlier symptoms, which were at the time ascribed to general paralysis, passed off so completely that his father and his wife believed that he had recovered, and requested that he should be discharged "well." He was so well that for three months he performed his duties as chaplain to the satisfaction of his friends, and went alone to Greece for a holiday, where he broke down for the second time. I think the very unfavourable conditions in which he was placed in the Corfu Maison de Santé had much to do with the rapidity with which the disease developed.

On re-admission, he had general exaltation, and this continued till the time of the onset of vomiting, which in his case took the place of the earlier fits; this was followed by loss of consciousness and death, the respirations becoming very slow before the end.

At the post-mortem we found very characteristic specific changes in the vessels, as well as adhesions about the frontal lobes, and the grey matter of the motor tracts was very pale. The case seems to be one of ordinary general paralysis, due to arterial diseases, and answers a question which is frequently raised as to whether there is anything special in the symptoms of cases of general paralysis depending on syphilis, and the answer is "No;" but that in the course of the disease, if syphilis be the cause, remissions are more perfect than is usual with the cases depending on other causes.

I have made use freely of the notes taken by Dr. R. Percy Smith.

CASE OF HÆMORRHAGE INTO THE MEDULLA OBLONGATA—RECOVERY.

BY J. W. BATTERHAM, M.B., B.S. (LOND.), F.R.C.S.

E. L., a single woman, aged 33, had suffered for nine years from spasmodic asthma, attacks of which were of almost daily occurrence.

She came under my care in October 1886, with slight bronchial catarrh. Her heart's action was irregular and somewhat feeble. There was no cardiac murmur.

On October 26, 1886, the patient, during a fit of coughing, felt a severe pain in her head and down the left side of her body, and became giddy. She then noticed that her left arm was "very weak," and her speech indistinct. There were no convulsions, nor did the patient lose consciousness.

She remained under my care for about a fortnight, and exhibited the following symptoms—

Paresis of *left arm and leg*, more marked in the arm, the movements of the fingers being most affected.

Slighter loss of power in the *right arm.*

Marked impairment of the movements of the *lips*, especially on the *right* side, the power of movement of upper part of face remaining unaffected.

Deviation of the *tongue* to the *left*, when protruded.

Great indistinctness of speech, from defective movement of tongue and lips.

Dilatation of *left* pupil. Temporary difficulty of accommodation.

Paresis of *right* side of velum palati.

Difficulty of deglutition.

There was no anæsthesia, and no impairment of sight, smell, hearing (taste not tested), nor was there any paralysis of extrinsic muscles of the eyeball.

During the first few days of her illness the patient complained of constant nausea, and vomited occasionally. She frequently experienced sensations of "falling" and of "sinking through the bed."

Frontal headache was present for a few days. Ophthalmoscopic appearances were normal. The temperature was normal. Urine also normal.

The patient was removed to the Hastings and St. Leonard's Hospital, where, after a temporary increase in the severity of her symptoms, she recovered in about two months.

During the greater part of her illness she was *entirely free from asthma;* but, as she recovered from her paralysis, the asthmatic paroxysms gradually reappeared, and, on her discharge from the hospital, were as frequent as ever.

Remarks.—In 751 cases of cerebral hæmorrhage collected by Gintrac, the medulla oblongata was only *twice* found to be the seat of the lesion.

In support of the diagnosis in the present instance I would remark—

(1) As to the pathological diagnosis, that the suddenness of the attack was indicative of embolism or hæmorrhage, the former condition being improbable in the absence of cardiac valvular disease, while the fact that the onset occurred during a violent fit of coughing pointed strongly to the latter condition as the cause of the paralysis.

(2) As to the localisation of the lesion, that a hæmorrhage, the occurrence of which is unaccompanied by convulsions, loss of consciousness or fall of temperature, and from the effects of which the patient recovers completely in two months, must necessarily be limited in extent.

For a lesion limited in extent to produce paralysis of widespread distribution on both sides of the body, it must be situate in a part of the encephalon where the motor tracts of opposite sides are closely approximated. It must be either in the pons or medulla.

The special symptoms of *pontine* lesion, viz. those of injury to its nerve nuclei, were absent; while the symptoms which were present are for the most part readily explained by the hypothesis of *bulbar* injury.

The facial paresis noticed was such as might be produced by implication of the *accessory* nucleus of the seventh nerve, which is situated near the nucleus of the hypoglossal, and is supposed to be concerned chiefly with the movement of the lips in speech.

The ingual, palatine, and pharyngeal pareses are indicative of injury to the hypoglossal and spinal accessory nuclei; the nausea, vomiting, and, presumably, the remarkable cessation of asthmatic paroxysms point to an interference with the vagal nuclei. The dilatation of the left pupil and temporary indistinctness of vision may have been due to irritation of cilio-spinal fibres. The "giddy feelings," "sensations of falling," "sinking through the bed," &c., are difficult of explanation unless we assume that the "inner auditory nucleus," with which the posterior root of auditory nerve is connected, has no auditory function, but receives those fibres of the auditory nerve which proceed from the vestibule, and which convey impressions giving rise, not to auditory perceptions, but to a "sense of direction." If such be the case, it is possible that an encroachment of the hæmorrhage on this so-called "auditory" nucleus may have originated the subjective sensations experienced by the patient.

I regret that I had no leisure to make anything like a complete investigation of this case, but trust that its rarity and its several points of interest may be an excuse for the publication of these scanty notes.

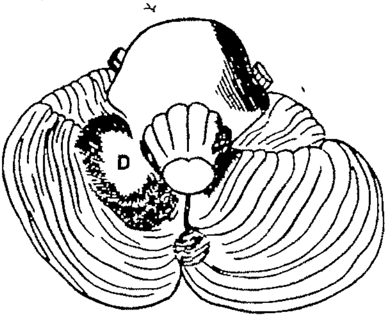
A CASE OF DEFECT IN DEVELOPMENT OF THE RIGHT LOBE OF THE CEREBELLUM, ASSOCIATED WITH DIMINUTION (?) OF THE OPPOSITE CEREBRAL HEMISPHERE.

BY PERCY FLEMMING, M.B., &c.

Demonstrator of Anatomy in University College, London.

THE brain, the subject of this note, was accidentally found in the dissecting-room, and in consequence there is no history attaching to it.

In viewing the base (see figure), a portion of the right hemisphere of the cerebellum is seen to be wanting, and the whole of that hemisphere is distinctly smaller than the left. More particularly, the deficiency is found to include almost the entire flocculus, the outer and anterior parts of the biventral lobe, and the adjacent part of the amygdala. The laminæ bounding the depression caused by the absence of these parts are all rounded off and covered by grey matter. In the floor of the depression is the white matter of the hemisphere, this being everywhere covered by pia mater.



D. Defect where the white fibres are exposed by the failure of development of the laminæ.

The left cerebral hemisphere appeared markedly smaller than the right, but it only weighed half an ounce less.

However, in view of the fact that Sir J. Crichton-Browne ('BRAIN,' Vol. I., p. 504; II., p. 42) found, in opposition to

Dr. Boyd, that the average excess of the right over the left cerebral hemisphere was very small, this difference of half an ounce assumes more importance than appears at first sight. The difference in the apparent size of the hemispheres was also more marked than the weight would suggest; perhaps there was also a difference in the specific gravity, but this point could not be determined.

Reviews and Notices of Books.

Anleitung beim Studium des Baues der Nervösen Centralorgane im gesunden und kranken Zustande. BY DR. HEINRICH OBERSTEINER, k. k. a. ö. Professor a. d. Universitat zu Wien. Leipzig u. Wien: Toeplitz u. Deuticke, 1888.

WE have great pleasure in bringing this work before the readers of "BRAIN," as we are confident that it will supply a want which is much felt, and it will not require any advocacy on our part to recommend it.

The book is of especial value, as it brings into juxtaposition the normal and pathological appearances of the minute structure of the nervous system, whereby the recognition of minute departures from health is much facilitated. The work is a pure study of the normal and minute pathological anatomy, and does not treat of the physiology or the clinical symptoms of diseases of the nervous system.

The book is divided into seven different sections.

In the first section, which treats of the methods of preparation of the various parts of the central nervous system, all the different modes of examining the tissue, such as teasing, hardening in the various reagents, the question of imbedding and cutting by various microtomes, are minutely discussed, and here we find Fol's modification of Fleming's method of staining the nuclei by perosmic acid and chromic acid, and a complete account of imbedding in celloidin.

Further on in this section the different ways of staining the nuclei, the medullated sheath and the axis-cylinder of the nerves are given, and especially Weigert's hæmatoxylin-copper method for staining the medullated fibres, and Pal's modification of the same, in which the hæmatoxylin is washed out with a solution of potassic permanganate, and one of oxalic acid and potassic sulphite; to this method Prof. Obersteiner gives the preference over the original one of Weigert, as it possesses the advantage of allowing the sections to be subsequently stained by carmine or eosine.

Weigert's method of fixing a series of sections between two layers of celloidin, and the corrosive sublimate process of Golgi, by which the various cells and their processes are so beautifully stained, are also minutely described.

Lastly, the other means by which the nervous system may be examined, viz. Flechsig's developmental method, Gudden's extirpation method, the mode of tracing the fibres by degeneration, caused by disease or operation, are discussed, as well as the evidence obtained from comparative anatomy and from experimental work.

It will be seen from the above that Professor Obersteiner has provided a very complete account of the various methods of examining the nervous system, including all the latest discoveries, and has thus furnished a concise reference, which will be invaluable to the practical worker in nervous histology.

The second section treats of the coarse anatomy of the nervous system, including the convolutions of the brain. This is illustrated by numerous original drawings, and we would call special attention to Figures 6 and 7, which are furnished with a numbered outline sketch to explain the various superficial parts of the posterior aspects of the medulla and pons. Here we would like to speak most highly of the way in which the whole work is illustrated. The drawings are nearly all original, and Professor Obersteiner has adopted the useful method of supplying an outlined numbered plan to accompany each illustration, instead of obscuring the original drawing with figures.

The third section treats of the histological elements of the nervous system.

Here, under the headings of nervous and non-nervous constituents, we have in the former list the nerve fibres and the nerve cells minutely described, as they are met with in health, and the condition in which they are found in disease; this includes the different stages of degeneration and regeneration of nerves, hypertrophied axis-cylinders, simple atrophy of nerve-cells, fatty and granular degeneration and calcification of ganglion cells, and the different conditions are illustrated by numerous drawings depicting the changes.

In the non-nervous constituents are included the vessels, epithelium lining cavities, and the supporting tissues, including connective tissue and neuroglia. In the description of the vessels, the perivascular and the pericellular lymph spaces are well shown, and Professor Obersteiner calls attention to the fact, that a lining membrane exists outside the adventitia of the cerebral arteries, from which numerous fine fibres radiate into the brain substance. He also remarks on the presence of pigment and fat granules on the normal adventitia of the small cerebral vessels, especially among children; but if the amount of fat granules be increased to a large extent, they must be looked upon as pathological.

The other changes in the vessels, such as calcification, changes in the intima, miliary aneurisms, and enlargement of the adventitial lymph space, are illustrated by original drawings, which show the actual condition better than can be described in words.

In the fourth section, in which the minute structure of the spinal cord is considered, the general arrangement of the connections of the different tracts of the cerebro-spinal system is detailed, and the different systems of Meynert, Flechsig, and Luys are enumerated. The different parts of the spinal cord are then described as regards its topographical and histological structure. Prof. Obersteiner calls in question the propriety of describing the anterior commissure of the cord as a true commissure, as it probably contains fibres which are decussating, and very few which connect parts having a similar character. At the end of this section the acute and chronic affections of the spinal cord are described, as far as their pathological changes are concerned.

In the fifth section, on the topographical survey of the brain, we have presented a drawing of the medulla, pons, and lateral ventricles, as seen from behind and above. The region between the lower end of the medulla and the anterior border of the corpora quadrigemina is marked off in this drawing by eighteen transverse lines, and for each of these lines a corresponding representation is given of a transverse section of the pons and medulla. We have therefore a series of eighteen transverse sections, which show every change in structure in the pons and medulla. The drawings are magnified to four times the original size, and in the region of the crura cerebri, a whole page is devoted to the representation; one half of the drawing is a most artistic picture, while the other half is drawn in outline, and contains the letters explaining the different parts. This arrangement is of inestimable value in helping the investigator to determine where to make a section on the medulla or pons, to ascertain exactly the condition of any given nerve nucleus—a task which is often exceedingly difficult. All the sections being so perfectly treated, it is not easy to draw attention to any particular one; but we might cite the sections showing the facial nerve, as an instance of the clearness with which its very difficult course is demonstrated. Probably no man has a better knowledge of the most intricate parts of the medulla and pons than Prof. Obersteiner, and he has treated this subject in a most masterly manner.

At the end of this section the complicated parts of the subthalamic region are described.

In the sixth section the various principal fibre-tracts are traced between the brain and the spinal cord, comprising the pyramidal tract, the posterior columns of the cord and the tracts in connection with it—namely, the lemniscus, the restiform body, and the other tracts of the cord, including the one first described by Gowers. These systems are illustrated by two diagrams, containing the probable course of the fibres. The different cranial nerves are then described very fully, and diagrams given to explain the deep origin of the 1st, 2nd, 5th, 7th, and 8th nerves. These diagrams are most useful, and convey more to the student than any amount of printed description could attempt to do, and they show at a glance the deep origin of these nerves according to the views of the present time.

It is impossible to discuss the way in which the individual nerves are treated, but suffice it to say that all the latest discoveries are embodied in the work.

The article on the cerebellum is of course very complete, as Prof. Obersteiner has largely contributed to the elucidation of its structure. We would call special attention to Fig. 151, which gives a representation of the protoplasmic processes of one of the Purkinje cells stained by Golgi's sublimate method, by which means an exceedingly dense and complicated network of fibres is seen to exist.

The structure and relation of the central grey masses of the brain are next discussed, including the optic thalamus with its complicated connections.

In the description of the corona radiata, amongst other things the fibres coming from the posterior part of the median convolutions and from the occipital convolution are described as going to the posterior part of the internal capsule, as well as to the thalamus, the outer corpus geniculatum and the anterior corpora quadrigemina.

The minute structure of the different convolutions of the cortex cerebri are next portrayed, and the fine network of medullated fibres of the cortex, as shown by Weigert's method, is illustrated by a drawing.

The work is concluded with sections on the membranes of the brain and cord and the large vessels.

It will be seen that this work is a most valuable contribution to the study of the anatomy and pathology of the nervous system, and we cannot speak too highly of the ability and skill which Prof. Obersteiner has brought to bear on this most difficult subject. We can confidently assert, that it will be an invaluable aid to all who are working at the pathology and anatomy of the nervous system, and we can only hope that its usefulness in this country will be further enhanced by its translation into English.

C. E. Beevor.

A Manual of the Diseases of the Nervous System.—By W. R. Gowers, M.D., F.R.S. 2 vols. 8vo, 463 and 975 pp. 370 figs. Churchill, London, 1886–1888.

The first instalment of this work, containing the Diseases of the Spinal Cord and Nerves, is no doubt already familiar to many of our readers; but we are glad that the publication of the second—more important, as well as voluminous—gives us an opportunity of noticing the work as a whole. We must, however, deny ourselves the pleasure of writing at any length concerning Dr. Gowers' volumes; for they belong to the category of works indispensable to those interested in Neurology, upon whose shelves they will speedily occupy a conspicuous position. Their contents, moreover, are so vast as to make it impossible in a review to enumerate the subjects handled by the author, far less to attempt an analysis and discussion of the views held by him on the numerous problems with which he has to deal. We shall limit ourselves, therefore, almost entirely to a statement of the leading features of this Manual, that characterise it as one of the very best published in any language.

What we admire, first, is the clearness of thought and language in the exposition, even in the more difficult portions of the subject. It is not every one who being a master, is at the same time a skilful expounder, and knows how to elucidate, whilst condensing, his theme. Secondly, we find the evidence, on every page of the book, of the author's individual familiarity with the topics he is discussing. Dr. Gowers' work in this respect often partakes of the interesting freshness of an original treatise, such is the wealth of personal information woven into his exposition of the subject in hand. Finally, we note the thorough mastery of the author of the most recent researches: a most creditable accomplishment when one considers the disproportion between the time at the disposal of a London physician in full practice, and the wealth of our contemporary neurological literature. Nor are the chapters devoted to the scientific or theoretical aspects of neuropathology the only ones deserving praise; those devoted to the practical aspect of the question, to nerve-therapeutics—this too often neglected "partie honteuse" of our branch of medicine—will be found if not always sanguine and cheerful, at least earnest and suggestive. Readers of French and German works on nervous diseases will however notice the larger share of attention given by Dr. Gowers, in common with most English authorities, to internal medication as compared with that bestowed upon hydrotherapeutics, massage, active and passive movements, and the like, under the various headings of treatment.

The Manual contains accounts not only of the leading morbid types, but of numerous minor forms of nerve disturbance, many of them to be found mentioned only in quite recent papers and monographs; and its interest and value are thereby enhanced for the professed neurologist, as much as for the student and general physician. Dr. Gowers would, however, add still further to our debt of gratitude to him as a guide through the field of neuropathology if, in his next edition, he were to give us a chapter of the pathology and treatment of stammering, a subject too much left to the "exploitage" of empirics. Morphinism might advantageously be adverted to under the heading of intoxications; and perhaps a sketch of the leading symptoms of general paralysis would be useful, especially in connection with certain phases of tabes. Another item which we miss in the present volumes is an index to the figures, many of which are very instructive. These suggestions, however, hardly deserve the name of criticisms. So in order not to derogate to the time-honoured tradition of reviewers always to find some fault with the work under notice, we shall attract Dr. Gowers' attention to some verbal mistakes in the course of the book. Thus, for instance, on p. 104 we find "Sepelli" for Seppili, "die" for "der" Complex; p. 924, the French expression for pleurosthotonos should be "arc de cercle latéral," &c. &c. But these are minor points. We find that Dr. Gowers adopts the absurd "pontine" for "pontal." Pontine is the name of marshes near Rome, but not the adjective derived from pons. Again, as the Greek "method" gives "methodic," so anŏd and kathŏd (usually misspelt and mispronounced anŏde and kathŏde) give anŏdic and kathŏdic, not anōdal and kathōdal. Again "faradaic," is not only pedantic but inexact: in forming an adjective from the name Faraday, the terminal diphthong *ay* should be replaced by the termination *ic*. With reference to the expression *myotatic*, introduced to designate the nature of the phenomena of tendon excitability known as knee-jerk, &c., we think (after some hesitation, we must confess) that it is a useful one, and deserving to be generally adopted.

A. DE WATTEVILLE.

Critical Digest.

CHOREA IN THE ADULT, AND IN THE OLD.

BY W. P. HERRINGHAM.

CHOREA is not a common disease in persons after thirty years of age, and the records of it are scanty; but the term is used of so many different classes of illness, that it is worth while to arrange and classify the cases at present recorded.

In the first place, elderly or old people are occasionally, though very rarely, attacked by a chorea which in course, duration, and result cannot be distinguished from the disorder so common in the young. Thus Roger[1] has recorded a case in which an old lady of 83 was affected by an ordinary chorea which came on without cause, and was cured in five weeks; Russell[2] writes of a woman of 77, who had it mainly on the left side, and recovered in three months; Sinkler gives a case of a man of 86 who recovered in a few months; Saundby[3] gives two cases, but the histories do not extend for a sufficient length of time to enable them to be classified. Ferguson[4] had a case in a woman of 74, who after much fatigue in nursing her husband, became somewhat irritable and excitable, and then began to twitch on the left side. She recovered in eleven weeks. In the Collective Investigation Report,[5] there are several cases which Dr. Mackenzie has grouped together as chorea at exceptional ages. No. 119, by Gowans, is a second attack of chorea following rheumatic fever in a man of 40. No. 223, by Alexander, is a woman of 63, in whom the attack lasted four months. She also had had it before. No. 222, by the same author, is a first attack in a woman of 68, lasting six months. Carline records a case, No. 180, of an old woman of 73, in whom a chorea lasted four weeks. She had had several attacks of late years. Aitken describes another case, No. 93, in a woman of 86, lasting one year. Of this kind also is Graves's case[1] of a man of 70, in whom the attack lasted several months, and Gauthier[2] records an attack in a woman of 75, due to peripheral irritation from extraction of a tooth, similar to those occasionally met with in the young. She recovered after fourteen days.

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[1] 'Gaz. d. Hôp.,' 1854, xxvii. 559.

[2] 'Med. T. and G.,' 1879, ii. 447.

[3] 'Lancet,' 1884, ii. 948.

[4] 'Lancet,' 1885, ii. 92.

[5] 'B. M. J.,' 1887, i. 425.

This class of case is like the chorea of the young, in that it is curable, and lasts a comparatively short though variable time, from a week or two to several months. There is not, however, if we may judge from so few cases, the same tendency to heart disease, nor is rheumatism so frequent an antecedent. In Nos. 119 and 180 of the Collective Investigation Report, and in one of Dr. Saundby's cases, the heart was diseased; in two it is not mentioned; and in Roger's, Russell's, Ferguson's, Gauthier's, and the remaining Collective Investigation cases it is normal. Acute rheumatism had occurred in No. 119, is not mentioned in four cases, and had not occurred in any of the others.

In the next class comes the chorea of coarse sub-cortical lesion, which often follows hemiplegia, sometimes precedes it, and sometimes is without it altogether. Whether, however, there be hemiplegia or no, the class is distinguished by possessing a coarse lesion, deeper than the cortex, and by limitation of the movements to the other side of the body. The lesion is usually a patch of softening, either, as originally described by Charcot, in, or pressing on the posterior part of the internal capsule behind the motor tract, or, as in the case of Gowers,[3] and apparently in that of Galvagni,[4] entirely within the optic thalamus; or again, as in the case of Gee,[5] in front of the motor tract of the internal capsule. It appears to me probable that Tuckwell's cases of plugging of the posterior and middle cerebral,[6] so often, but I must think wrongly, quoted in support of the theory which ascribes chorea to capillary embolism, are really to be placed in this class.

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[1] 'Clin. Med.,' i. 537.

[2] 'J. de Méd. et Chir. Prat.,' 1881, lii. 66.

[3] 'BRAIN,' Vol. I. p. 57.

[4] 'Riv. Clin. d. Bologna,' 1880, x. 268.

[5] 'St. Barth. Hosp. Rep.,' xvii. 285; and 'Brit. and For. Med. Chi. Rev.,' 1867.

[6] 'Brit. and For. Med. Chi. Rev.,' 1867.

A third class is formed by a disease of which there are few notices, in which many members of a family are attacked by chorea in adult, or middle life, which lasts till death, and is usually accompanied by insanity. Huntington[1] was the first to describe such cases. I am unable to find his paper in London, and therefore rely on the account of it given by Wharton Sinkler.[2] Huntington himself, his father, and grandfather, knew some families in Long Island in whom this form had been under observation for many years. It came on gradually, after middle life, and affected males more than females. In most cases, insanity and a tendency to suicide supervened. Ewald[3] writes of a mother and two daughters in whom an incurable chorea began at or after thirty, and also of another family in which nine individuals of three generations were similarly afflicted. Peretti[4] gives a family-tree, showing many individuals in three generations affected in middle life with chorea, a considerable proportion of whom were also insane. King[5] records another family, in three generations of which the disease has appeared, and gives a good description of the condition as he has observed it. These cases began about thirty years of age, were always associated with insanity, and showed a peculiar gait. West, in the Collective Investigation Report,[6] describes another family similarly affected as regards movements, but does not mention insanity in connection with it. There have been no post-mortems recorded of this disease.

The remaining or fourth class of cases differ from the first class by being chronic, and incurable; they differ from the second, in that when a lesion has been found, it has been such as to affect the cortex rather than the deeper parts; and they differ from the third in the absence of any hereditary tendency.

[1] 'Philad. Med. and Surg. Reporter,' 1872, Ap. 13.

[2] Art. "Chorea" in Pepper's 'System of Med.,' vol. 5, 440.

[3] 'Ztschft. f. Klin. Med.,' vii. Supplement H. p. 51.

[4] Peretti, 'Berl. Kl. Woch.' 1885, No. 52.

[5] 'N. Y. Med. Journ.,' 1885, i. 468.

[6] Loc. cit.

This class is divisible into two groups: (1) of cases preceded or accompanied by mania or dementia; (2) of cases with no mental alteration. Of the first group, Hale[1] records the history of a coachman, aged 65, who had chorea for six years, had two attacks of mania during that time, was very intemperate, very irritable, and died of acute bronchitis. He had cerebral pachymeningitis, and his brain was shrunken, very pale, and hard. Maclaren[2] relates the case of a man of 36, in whom the chorea had begun seven years before, after severe frontal headache. He became childish, and died. He had subarachnoid hæmorrhage over both hemispheres for a considerable area, and general atrophy of the convolutions, with a small spot of softening in the right optic thalamus. The movements were general, not confined to the left side, and therefore probably not to be explained by the disease of the thalamus. Mackenzie Bacon[3] describes a woman of 58, in whom the chorea lasted four years, and was combined with mania. Dissection showed chronic arachnitis, a shrunken brain, and a large collection of serum in the ventricles. Macleod[4] gives three cases. The first is that of a man aged 52, in whom chorea lasted six years; dementia ensued, and he died in coma. He had a subdural hæmorrhagic cyst over both motor areas, and the convolutions were atrophied. The second is a woman aged 62, who had chorea for six years, gradually became demented, and had a similar subdural cyst over the left motor area, with a thick and adherent pia mater. The movements were general. The third is her sister, aged 72, who died in stupor after three years' chorea, and who had several hard tumours in the dura mater. Devis[5] describes an old man of 79 with dementia, who, while in hospital for a broken thigh, developed left-sided chorea and died, but was not dissected. Berkley[6] gives a long and careful account of the microscopic examination in the case of a woman of 41, who seven years ago developed chorea five hours after being severely frightened by a murder committed before her. She gradually became demented, and died. He found in the brain

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[1] 'Path. Trans.,' v. 16.

[2] 'Journ. Ment. Sci.,' xx. 97.

[3] 'Journ. Ment. Sci.,' xxvi. 258.

[4] 'Journ. Ment. Sci.,' xxvii. 194.

[5] 'Med. T. and G.,' 1879, ii. 446.

[6] 'Philad. Med. News,' xliii. 1883, 200.

"dilatation and thickening of arterioles, small gangrenous spots round many vessels; amyloid corpuscles in the lymph sheath; vacuoles in white and grey matter; varicosities of the sheath of Schwann, and absence of axis-cylinders in the nerve fibres; and complete pigmentary degeneration of a large number of the cortical and ganglionic cells, with loss of the prolongations." There were similar changes in the cord, the cellular degeneration being especially marked in the enlargements, while there was considerable antero-lateral sclerosis in the remainder of the cord. The heart was fatty, but without a trace of past or present endocarditis. Bevan Lewis[1] relates the case of a man, aged 56, who had general choreic movements after a paraplegia of the legs six years ago, and who had some mania six months before death. There was congestion of the meninges of the brain, with a granular degeneration throughout the motor cells of the cord, but the cortex of both brain and cerebellum was quite normal to the microscope, and there was nothing amiss in the basal ganglia.

It is of interest to compare with this group Case No. 365 of the Collective Investigation Report, also by Macleod, in which a woman, aged 40, was seized with a severe chorea immediately after blows on the head. There was mania for a month, and the chorea lasted five months. Macleod suggests a lesion in the front of the brain, evidently likening it to the fatal cases he had before recorded.

Of the second group, that in which no dementia or insanity exists, Russell[2] describes a case which had lasted over eighteen months, and was unrelieved by treatment, the patient being a man of 68. Sinkler[3] mentions another in a woman of 82. McLearn[4] relates a third in a man of 56, in which there was the subjective feeling of heat so common in paralysis agitans. None of these died under observation. Dickinson[5] had a case in a woman of 54, who died of granular kidneys. Nothing was found post mortem to explain the chorea. I had a case among my out-patients at the West London Hospital in a man of 68, who had had the disease for fifteen years. It affected only the right arm and the head, which was continually being jerked to the right side, that is, to the side affected. Although so confined, it was violent. A continually increasing anæmia attacked him, for which he was admitted into the hospital under Dr. Donald Hood, and of which he died. I examined the brain carefully, and could find no abnormality in it whatever, but I regret to say that I did not make a microscopic examination.

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[1] 'Journ. Ment Sci.,' xxx. 256.

[3] Loc cit.

[2] 'Med. T and G.,' 1878, ii. 627.

[4] 'Lancet,' 1885, i. 337.

[5] 'Med. Chi. Trans,' lix. 1.

A remarkable case is related by Wood,[1] in which a man of 74 suffered amputation of the left thigh for senile gangrene, and five years later began to be affected with irregular clonic spasm, mingled with some tonic spasm, called by Wood "chorea of the stump," which afterwards spread to the arm and face of the same side. There was never any pain connected with the chorea, nor did any kind of peripheral irritation increase it.

Charcot, speaking of the two groups of this class, remarks that in the few cases he has dissected, there has been no endocarditis, and that there is no connection with rheumatism. This is borne out by the other observers who have examined cases after death. I have mislaid the post-mortem notes of my case, but I do not remember any heart disease being found, and there was no murmur during life. The man had never had any previous illness.

It is worth noting that the only cases in which gross disease has been found have been in the insane. That these, however, are not constant in the brain, even in this group, is shown by the results of Bevan Lewis's examination. It is too early to lay any stress upon the changes hitherto recorded. All we can do is to classify the cases according to their clinical symptoms. To fulfil this task is the object of the present paper.

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[1] 'Philad. Med. Times,' Nov. 8, 1879, quoted in 'Med. T. and G.,' 1880, i. 16.

Abstracts of British and Foreign Journals.

Pitres et Vaillard on Acute Ascending Paralysis (*Arch. de Physiol. Norm. et Path.*, 1887, p. 149).—The case was one of a man, æt. 22, convalescent from typhoid fever. He woke up one morning and found that his legs were paralysed and anæsthetic, with loss of reflexes. During the day the paralysis spread upwards, involving the abdominal muscles; later on, the thorax and arms were paralysed. Respiration became diaphragmatic, aided by the shoulder and neck muscles. Bladder and rectum paralysed. Deglutition not affected. Death in about 12 hours.

Post-mortem.—The central nervous system was quite free from disease. The nerves of the upper and lower extremities showed signs of disease as follows:

(*a*) Fibres atrophied to such an extent as to show only their primitive sheath with *débris* stained slightly by the osmic acid.

(*b*) Fibres in which the myeline was finely broken up and gathered into varicosities.

(*c*) Fibres in which the myeline was divided into larger masses. The axis cylinder was affected in a similar manner.

This description applies to such nerves as the post-tibial, sciatic, musculo-cutaneous, radial, &c. The spinal roots were affected to a much less degree.

The authors consider that this case throws some light on that mysterious disease described and named after Landry. They discuss the opinions of other writers at some length, and show that while a few have described change in the cord, a considerable number have shown that the central nervous system is free from disease, and several have described changes in the peripheral nerves.

Letulle on Mercurial Paralysis (*Arch. de Physiol. Norm. et Path.*, 1887, p. 301).—The author begins by noticing that the attention of most writers is directed chiefly to the tremors of mercurialism, and that the pareses have been passed over by the larger number of them. He shows that paresis, general or partial, is exceedingly common, and may be almost the only symptom. Anæsthesia and even patches of hyperanæsthesia are very common. Pain in the affected limbs is rare. There is never any atrophy of muscles. The reflexes are increased in cases in which there is much tremor, but diminished often in the paretic cases.

The upper limbs are much more often affected than the lower. The trunk is rarely affected. He draws his cases from the mines of Almaden, and from the felt-makers in Paris.

But in order to study the effect of the poison on the nerves he subjected rats and guinea-pigs to as long a course of mercurial treatment as possible. His experiments were of three classes. (1) Injection of mercurial peptone along the course of a nerve. (2) Injection of the same anywhere in the body. (3) Inhalation of mercurial vapours.

In most cases he got a definite affection of the muscular nerves. This affection was apparently confined to the myeline sheath of the nerves, and therefore differs considerably from the ordinary parenchymate neuritis. It consisted of (1) swelling granulation of the myeline; (2) granular disintegration, followed latterly by (3) atrophy of the myeline. This condition obviously differs from other forms of neuritis, in the fact that only the myeline sheath is affected, the axis cylinder being spared indefinitely. This will explain the great rarity of atrophy in hydrargyrism, but it is difficult to explain the paresis, at least by the ordinarily accepted views of the functions of the myeline sheath.

The paper is well illustrated by cases and details of experiments. H. H. Tooth, M.D.

Chauffard on Sudden Blindness in Lesions of the two Occipital Lobes (*Revue de Médecine*, Feb. 1888).—It is not easy to refer sensory functions to their place in the cerebral cortex, for not only are there few clinical facts to guide us, but experiments on the lower animals are open to obvious difficulties. The localisation of sensory thus affords a great contrast to the localisation of motor functions, which indeed may almost be regarded as a finished work.

In respect to Sight, it has been proved by Allen, Starr, Seguin, and Exner, that bilateral corresponding *hemianopsia* results, without any other sensory or motor symptom, from destruction of part of the occipital lobe. Cases have also been recorded in which in General Paralysis *total blindness* has been noted corresponding with lesions of both occipital lobes. Pflüger relates that a young man received the discharge of a gun in the occipital region, causing immediate blindness and ultimately double optic neuritis.

He died, and at the autopsy grains of lead were found scattered in both occipital lobes.

The author gives four cases, three of which are taken from Berger and Bouveret, in which sudden complete blindness was associated with lesions of these lobes. In his own case, that of an old man of seventy-four, there were three hæmorrhages, one, on the right side, occupying the greater part of the occipital lobe; and two in the left hemisphere. One of these lay in the external capsule, but the larger clot was contiguous to the *pulvinar*, at the very point where, according to Wernicke and others, the fasciculi of the optic tract leave the central ganglia. From this point they pass outside the hinder limb of the lateral ventricle, and finally reach that portion of the cortex which forms the cuneiform lobe and first occipital convolution. In the three other cases, also old men, the occipital regions were affected by softening, from thrombus or embolism. This association of symptoms and lesions accords well with the experimental results obtained in animals by Ferrier and Yeo.

Dr. Chauffard considers that in his case, as in others, simple hemianopsia must have preceded the total loss of sight. Hemianopsia may be often latent or undiscovered. Blindness is thus the result of two seizures on two separate occasions. After the first seizure (hæmorrhage in the left hemisphere) the uninjured centre doubtless took on by substitution more or less of the functions of the other. After the second hæmorrhage (in the right hemisphere), substitution became impossible. It is quite exceptional for the two occipital lobes to be attacked at once. The question arises whether hemianopsia, due to cortical lesion, tends by extension to the companion centre to pass into anopsia. If so, the symptom is one of serious import; for, according to the present author, cases of cortical anopsia invariably prove fatal within a short time. We may note, finally, as diagnostic features, that the blindness is complete and sudden, that "optical memory" is retained, that the fundi are normal, and, what is important, that the pupils are equal, moderately *dilated* (not contracted as in uræmic intoxication), and preserve their reflex excitability to light.

Spillman and Parisot on Peripheral Injury and Tabes (*Revue de Médecine*, March 1888).—In answer to the enquiries, "Can peripheral injuries be the starting-point of Tabes? Are they a sufficient cause?" the authors relate a series of cases in which locomotor ataxy followed the infliction of various injuries. It is not difficult to believe *à priori* that even a small lesion of a part of the sensory system might lead to an affection generalised over the whole of that system; although it is impossible to assert without evidence from morbid anatomy, that is not at present forthcoming, *how* that generalisation is effected. But, in their opinion, clinical evidence establishes the fact.

The interval between the injury and the first symptoms of tabes varied from a few weeks to as much as eighteen years; and it is worthy of note that, in five out of the fourteen cases quoted, nervous heredity, alcoholism or syphilis were stated to be present. Former writers have noticed an occasional connection between injuries and locomotor ataxy; and Erb (quoting Schulze) cites cases in which it has followed fracture of the thigh or a fall on the abdomen. Petit considers that wounds at a distance "hasten its development," and others have counted *injuries* in a general way as among the causes of tabes. The present writers carry this view much further. Given a personal or hereditary predisposition to nervous affection, they hold that, besides fractures, many slight injuries, such as sprains or wounds of the feet, or even operations in dentistry or for cataract, may be sufficient determining causes. Again, the first symptom of the tabes arises always, in their view, in the injured region. After injury to the left foot, the symptoms commenced after five months with lightning pains in the same limb; after contusion of the abdomen, with gastric crises, two months from the date of injury. This circumstance will often afford a guide in the history of each case to the original exciting cause. Finally it is allowed that diatheses—alcoholism, syphilis or rheumatism, may exert in some of these cases a predisposing influence.

FORTESCUE FOX, M.D.

Raymond on Sweating of the Face (*Arch. de Neurol.*, Jan. 1888).—The first case recorded is that of a man, aged 31, who noticed in 1880 that sweating of the right side of the face always appeared two or three minutes after he had taken food, and ceased when he left off eating. The sweating extended on to the neck and as far as the shoulder. Hot food induced it most rapidly; movements and the idea of eating had no effect; all other symptoms were absent, except that the right pupil was dilated and not affected by light, and below the right wrist there was difficulty in distinguishing between hot and cold objects. Injections of two milligrammes of pilocarpin produced a local sweating of the face on the right. Neither galvanism, nor atropine, nor ergotine, nor tannin produced any effect on the patient's malady.

The author divides the cases of local sweating into the following groups. First:—Those in which there is an alteration in the cerebro-spinal nervous system. Second:—Those in which the cervical sympathetic, or the first thoracic ganglion is affected. As a rule, in these cases, the pupil is contracted, but one is cited in which it was dilated. In this case there was chronic inflammation of the superior cervical ganglion on the same side. The author's second case is placed in this second group; it is that of a man, aged 39, who had phthisis; he also had unilateral right-sided sweating, especially of the forehead. There were no vaso-motor or pupillary symptoms. He died suddenly, three days after an epileptiform attack. The tubercular pleurisy had bound down and caused chronic inflammatory change in the right inferior cervical ganglion. Third:—Those in which the nerves of the face are affected. Fourth:—Those in which the sweating is reflex. These cases usually occur in nervous hysterical people. Many examples of this group are given, such as that of a young man in whom facial sweating was induced by smelling vinegar.

Thus there are two classes of cases characterised by increased sweating; those in which there are and those in which there are not vaso-motor troubles. The lesion in the former is in the sympathetic of the neck; the fact that the pupil is sometimes dilated and sometimes contracted depends upon the fact, that the pupillary and vaso-motor nerves are probably distinct, and one set may be stimulated whilst the other is paralysed. The author then gives an account of the various chronic inflammatory changes that have been found in the superior cervical ganglion, and concludes that these irritate the sweat-secreting nerves. Sometimes these changes exist without there being any alteration of sweating. Lastly, he points out, that the pupillary changes are permanent whilst the sweat ones are transitory; that the increased secretion of sweat may extend beyond the face, so that treatment has very little effect.

HALE WHITE, M.D.

BRAIN.

JULY, 1888.

Original Articles.

ON THE FUNCTIONS OF THE TEMPORAL AND OCCIPITAL LOBES: A REPLY TO DR. FERRIER.

(With two Plates.)

BY E. A. SCHÄFER, F.R.S.

I SHOULD have been content to pass Dr. Ferrier's polemical article in the last number of 'BRAIN' in silence,[1] were it not that the author directly contravenes the accuracy of the descriptions which I have given of the post-mortem condition of certain of the brains which had been operated on by Dr. Sanger Brown and myself, and indirectly accuses me of falsifying the representations which I have given in my paper:—"But Professor Schäfer's figures (Figs. 5 and 6) of the brains of these monkeys must be regarded more as a sort of pictorial representation of his own idea, or wish, as to the extent of the lesions, rather than of the reality. Having myself examined both preparations, I most emphatically deny the accuracy of Professor Schäfer's diagrams. In neither the one nor the other were the superior temporal gyri completely destroyed: considerable portions remaining absolutely intact in both hemispheres, and notably so in the brain which Fig. 6 professes to represent."

Now it happens fortunately that the brains in question are still existent. It also happens that, in spite of the fact that they have been sliced in order that the depth and extent of the lesions might be more accurately gauged, they are capable of being pieced together and photographed so as to show accurately what is really the extent of the lesion in each case. And from the photographs, which I here reproduce (Figs. 1 and 2), it is clear that Dr. Ferrier (whether owing to the fact, that the examination which he made was hasty and superficial, or whether he was unconsciously influenced by preconceived opinions) has committed a serious inaccuracy, in stating that "considerable portions of the superior temporal gyri remain absolutely intact in both hemispheres, and notably so in the brain which Fig. 6 professes to represent."

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[1] "Schafer on the Temporal and Occipital Lobes," by David Ferrier, M.D., F.R.S.: 'BRAIN,' April 1888.

Before I allude to the photographs at greater length, I may be permitted to spare the reader the trouble of referring back to the original paper, by reproducing the paragraph in which I have described the characters of the lesions :—

"In six monkeys we have more or less completely destroyed the superior temporal gyrus on both sides. I say more or less completely, because *in one or two a small shred of grey matter belonging to this convolution was found post mortem*, but practically the lesion was complete in all six, some of the grey matter within the fissures bounding the gyrus being all that could be taken to represent the convolution, and even this *being deprived of its medullary centre*. . . . But in order to make assurance doubly sure, we in one monkey . . . separated up the fissures bounding the gyrus and scooped it out entirely from the very bottom of the fissures, so that not a trace of the convolution in question should remain (Fig. 5)."

Now, except for this brain, it will be seen that I have not stated that every trace of the superior temporal gyrus has been removed in the six instances quoted, nor is it so represented in my diagrams. On referring, for instance, to Fig. 6, it will be seen that a very narrow strip of the gyrus lying next the Sylvian fissure is represented as having been left. But even this might in a diagram (which is all that the illustrations profess to be) with complete justice have been represented as being removed, for *as the sections show* it is only a shred of grey matter in the depth of the fissure that is left, the rest of the grey matter and the white matter belonging to it having been completely removed along with the remainder of the gyrus and even the remainder of the lobe.

How complete the removal has been is so evident from the accompanying photographs (Figs. 1 *a* and 1 *b* from the two sides, and Figs. 1 *c*, 1 *d*, 1 *e*, 1 *f*, and 1 *g* in section, the sections passing vertically through the lesion, at various points from behind forwards) as to require no words of mine to accentuate.

On the other hand, I have distinctly stated that in the brain represented in Fig. 5 precautions were taken to remove every trace of the superior temporal convolution on both sides.

Dr. Ferrier, on the other hand, boldly asserts that "considerable portions remain absolutely intact in both hemispheres." What do the photographs show?

An observer unskilled in the examination of cerebral lesions of this kind would, I think, come to the same conclusion as Dr. Ferrier has done, from the superficial examination to which alone he appears to have subjected this brain. There is certainly, especially well marked upon the right side (see the photograph, Fig. 2 *a*), a convolution or what appears to be such, exactly in the situation of the superior temporal, although somewhat less prominent than that well-marked gyrus. But if we observe the sections (Figs. 2 *c*, *d*, *e*) carefully, the convolution in question turns out to be the island of Reil! The superior temporal gyrus has in truth disappeared, and, as is frequently the case with these artificial cerebral lesions, the underlying parts have tended to be protruded towards the surface. Only at the extreme tip of the lobe is there a shred of grey matter left belonging to this gyrus, and that shred is devoid of medullary centre (Fig. 2 *e*, on the right side of the section).[1]

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[1] The diagrams which I gave in my paper, although only outlines, accurately represent the superficial extent of the lesion in each case. The paper itself being merely a résumé of a longer and more detailed account of the experiments, the joint production of Dr. Sanger Brown and myself, which was read before the Royal Society on Dec. 15, 1887, and is shortly to appear in the 'Philosophical Transactions,' I did not judge it to be necessary to illustrate the abstract by more elaborate drawings. All the figures which Dr. Brown and I have published of the brains which we have operated on (and the same remark applies to those operated on by Mr. Horsley and myself) have been executed with the greatest care and fidelity to nature by my assistant Mr. E. P France, who has in every case first traced out upon a glass plate placed over the brain all the outlines of the convolutions and the exact extent of the lesion, and has then transferred the tracing to paper and elaborated the drawing By this

Having been forced, sorely against my inclination, to take up my pen in vindication of the accuracy of the facts which had been impugned by Dr. Ferrier, I may perhaps be permitted to indulge in a short notice of one or two of the other points which my critic has raised. And first let me notice that he evades the main argument regarding the superior temporal convolution, and endeavours to conceal the evasion, by raising a side issue as to whether there is any shred of the grey matter of that gyrus remaining in any of the cases recorded by Dr. Sanger Brown and myself. My argument was this:—In a *single instance* recorded by Dr. Ferrier (in conjunction with Professor Yeo) what purported to be complete destruction of the superior temporal gyrus on both sides[1] was followed by complete loss of hearing, which lasted for thirteen months ("*manifestly totally deaf*" are the words used[2]). In *six cases* recorded by us, in every one of which the removal was as complete as—probably more complete than—in the monkey described by Ferrier and Yeo, hearing was not only not permanently abolished, it was not permanently affected so far as it was possible to determine in monkeys. In one at least of these six cases the convolution in question was not merely cauterised, but was radically removed on both sides with exactly the same result. What, then, is the natural inference

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means a representation is obtained as faithful as any photograph, and far clearer; because it commonly happens, when the superficial grey matter only has been removed, that the subjacent cerebral substance becomes pushed up towards the surface, and the original depression is thus obliterated. Thus there is a want of the necessary contrast of light and shadow requisite for a clear photograph, and it is on this account that so little can be made out regarding the extent of lesion in many of the photographs which illustrate the paper by Drs. Ferrier and Yeo, in the 'Philosophical Transactions' for 1884 In the two cases represented in this paper, however, I have easily succeeded in obtaining clear photographs, because the lesion has been so profound as to leave a considerable gap, in spite of a certain amount of pushing up of the subjacent cerebral substance which has occurred.

[1] I here reproduce (Figs. 3 *a* and 3 *b*) Dr. Ferrier's photographic representations of the brain of the monkey which he described as "totally deaf," in order that they may be compared with ours, and because they plainly show that the destruction of the superior temporal gyrus was the reverse of complete. This is still more obvious in the sections ('Phil. Trans.' 1884, Plate 22, Figs 25 to 38) which show that upon the right side nearly half the convolution remains untouched.

[2] Ferrier and Yeo, p 506 The italics are mine.

to be drawn from a comparison of these results, but that the auditory faculty is not *localised* in the superior temporal gyrus?[1]

Now this position is not touched by any of the arguments which Dr. Ferrier has brought forward. His statement, that in our experiments considerable portions of this convolution have been left, the photographs clearly show to be erroneous, at least for the two cases which have been directly called in question, and it would be not less erroneous for the other four cases which we brought before the Royal Society. And even if it be admitted, that shreds of grey matter have been left here and there, they do not in any case amount to as much as remained in the case of the monkey which Dr. Ferrier supposed to be deaf: this we may gather both from the authors' own admission as well as from the photographs they give both of the whole brain (which I have reproduced) and of sections through the lesion. Dr. Ferrier's other arguments consist, in the first place, of an appeal *ad misericordiam*, to the effect that if we do not localise the auditory sense in the temporal lobe (and preferably in its superior gyrus) there is nowhere else to put it, all the remaining parts of the brain being already tenanted; and, in the second place, of the citation of two clinical and pathological records, which are introduced, with a blast of trumpet, as "cases of transcendent importance."

One of these, reported by Wernicke and Friedländer (in 'Fortschritte der Medicin,' 1883, p. 177), was of a syphilitic growth in each hemisphere, in a woman, long the subject of epilepsy, and later on of hemiplegic attacks on both sides. Other symptoms appeared gradually, including deafness, imperfection of speech, and general stupidity, so that the patient was looked upon as of aberrant intellect.

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[1] When we find a statement made to the effect, that a function is *localised* in a particular part, the statement, if it means anything at all, surely implies that it is connected with that part to the exclusion of others. This is the sense in which the expression has been used in describing the localisation of functions in the brain, and of this no one can be better aware than Dr. Ferrier himself. In this sense Dr Ferrier has endeavoured to prove that the *cerebral auditory faculties are localised in the superior temporal gyri*. But if it is shown, even in a single instance, that these gyri can be entirely removed without loss of hearing, it is certain, whatever may be the amount of negative evidence brought forward on the other side, that those gyri cannot be the sole seat of auditory perceptions; in other and looser phraseology, cannot be *the* auditory centre. The case is put very clearly and forcibly by the late George Henry Lewes, in a review of the first edition of the "Functions of the Brain" in 'Nature,' 1876: "Whenever a function persists or reappears after the destruction of an organ, this is absolutely conclusive against its being the function of *that* organ."

Although he professes to give the particulars of this case at some length, all that Dr. Ferrier says about the condition of the brain is that "an extensive lesion was found in each temporal lobe, invading the superior temporal convolution on both sides (see figures accompanying paper). The rest of the brain exhibited no abnormality." On referring to these figures and to the authors' description, I find the conditions to be very different from that which the above account would lead one to suppose. In the first place, the lesion (a gummatous infiltration) is not symmetrical on the two sides. On the left side it involves much more of the temporal lobe than on the right, but even in the former the anterior third, if not the anterior half, of the lobe is normal. On the right side the tumour, equal to about a shilling in diameter, occupies the junction of the parietal, occipital, and temporal lobes, but involves very little indeed of the latter, and hardly any of the superior convolution. But the authors state that the new formation extended on both sides in the depth, so as to embrace the part of the corona radiata with which the temporal lobe is connected, and they are of opinion that (not the superior convolution only, but) the whole lobe was thereby put out of function by it. (No figures illustrating the depth of the infiltration accompany the paper.)

The authors infer that the temporal lobes are the central seats of the auditory sense. Dr. Ferrier, however, would go further, and say that this case is "entirely in harmony with my own more strict localisation of the auditory centre in the superior convolution." One would have felt grateful had Dr. Ferrier pointed out how this "harmony" is maintained. I at least fail to see the connection! For as we have just seen, *not the superior convolution merely, but the whole temporal lobe* was cut off by the tumour from its deeper connections; and it is certain, from the mental condition of the patient and the pareses which were observed, that parts of the brain other than those which were the actual seat of or were cut off by the new growth must have been involved.

The other case is one reported by Dr. Shaw, who himself is careful to remark concerning it, that "it is not strong evidence of this localisation of centres of hearing and vision," and is otherwise very guarded in the inferences he draws from it. Even Dr. Ferrier regards it as "not being absolutely conclusive." Under these circumstances I may perhaps be excused treating it as a serious item in the argument.

"Cases of transcendent importance" appear to be rare, and Dr. Ferrier must fall back upon his experiments on monkeys. And upon these he has thrown a flood of light by the publication of the notes which he gives at pp. 13 to 16 of his article. To any impartial mind these show conclusively, that the monkey which was reported in the 'Philosophical Transactions' as having been for thirteen months "manifestly totally deaf," was far removed from that condition. "Animal occasionally turned its head" (towards sounds). "Occasionally it turned its head coincidently with the noise." "Every now and then doubt seems raised by the animal looking at the same time a noise is made." "More than doubtful." "Signs of reaction to sounds are less evident" (this was a month after the operation; they were therefore evident before). "Occasionally it is judged to hear." "Occasionally, as before, there was an appearance of a start or a look round apparently conditioned by the noise" (this was thirteen months after). It is unfortunate that these remarks were not published at the time the case was brought before the notice of the physiological world. They would have permitted conclusions to be drawn other than those arrived at by the authors, and we should have had one error less to eradicate from our notions regarding the functions of the brain.

Dr. Ferrier (pp. 9 and 10) draws attention to "the difficulties that have to be encountered, and the fallacies that are apt to be committed, in investigating the sense of hearing in the lower animals. Care must be taken to discriminate between the sense of hearing proper, and mere reflex reaction to sudden, sharp, or loud sounds, a reaction not incompatible with real deafness. Mere coincidences in animals keenly on the alert to everything going on around them, must be scrupulously eliminated. The tests of hearing should be such as to entirely avoid attracting the animal's attention by other channels of perception, such as sight, imitation of other animals, a sense of proximity conditioned by vibration, agitation of the air, and such like."

We can now see by these notes that he has given to us how he has himself carried out his precepts. For we read that this identical animal "pays no attention to sounds—*tapping, scratching, &c.—on side of cage*,[1] which alarm its companion towards either side. This was carried on repeatedly for the space of an hour without eliciting any sign of perception."[2] "July 5th. Made several experiments with electric bell *placed against side of cage*." "July 7th. I made repeatedly a *scraping sound on floor* with foot as if going up to cage." "Dec. 12th. Does not pay any attention to *scratching on cage*." The animal therefore did not respond to *vibrations which must have been transmitted to the parts of the skin in contact with the cage!* Why has not Dr. Ferrier localised cutaneous perception also in this gyrus? We read that on "March 31st. While sitting to-day having its back scratched a pistol cap was exploded close to cage: not the slightest start, though the others and Mr. N., who was scratching the monkey, made a sudden start." Cannot Dr. Ferrier see that he is trying to prove too much? Mr. N. is scratching a monkey and suddenly starts, but even under these trying circumstances this remarkable monkey remains absolutely quiescent! Dr. Ferrier is capable of being amused (p. 17, line 1) at my comments on the above record;[3] but I think he need hardly have gone so far to seek amusement: there is plenty to be obtained from the record itself. But Dr. Ferrier tells us that he does not rely upon this one case alone; there are others from his earlier experiments (recorded in the 'Philosophical Transactions' for 1875) which he regards as corroborative of this one, if not of equal importance in the chain of evidence for his localisation of hearing. I can only express surprise that even their author should continue to attach the least importance to the experiments upon the special senses which are recorded in the paper referred to. So far as I know, not a single physiologist who has repeated them has been able to corroborate Dr. Ferrier's results, or to accept the conclusions which he was led to deduce from them. What scientific value can be attached to statements regarding the apparent absence of certain perceptions in animals which have hardly recovered from the stupor of an anæsthetic drug, or in which active inflammatory processes are going on in the cerebral substance? The danger of drawing conclusions from experiments made under these conditions is obvious, and is in no way better exemplified than in the fact, that their author has found it convenient, in certain cases, to rely upon their crudity, and tacitly to abandon positions which, trusting to their results, he had deliberately assumed.[1]

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[1] The italics are mine.

[2] We are not surprised to hear that afterwards, "as soon as I come within sight, it looks interested."

[3] I here desire to enter a protest against remarks being attributed to me which convey an entirely erroneous impression of my argument. Dr. Ferrier must be aware that to publish statements as having been made in any discussion before a scientific society, of which no short-hand report has been taken, and without having previously submitted them to the person to whom they are attributed, is a most unusual proceeding, and one which is easily capable of creating a false impression of the opinions enunciated; and I regret that he has not hesitated, for the sake of endeavouring to secure a momentary argumentative triumph, to break through the salutary rule which prohibits such irregular publication. His adoption of this course necessitates my here recording what it was I really did

Feeling to all appearance the weakness of his case for the localisation of all auditory perceptions in the superior tem-

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say in the few remarks which I made at the meeting referred to, in contrast to what has been attributed to me. Dr. Ferrier states that, after hearing the above notes respecting his monkey, I suggested first that they showed that the animal was not really deaf, and then, "almost in the same breath," suggested that the monkey was stone-deaf, not only after but before the operation. In making this statement, Dr. Ferrier has perverted both the order and the sense of my remarks. *Before the notes were read* I said that our six cases proved to demonstration, that if Dr. Ferrier's monkey were really stone-deaf after the operation (which I did not myself believe to be the case) he must by accident have lighted upon a monkey which was stone-deaf before the operation. *After the notes were read* I said that it must be perfectly clear to all who heard them, that the monkey was not really deaf at all. And I think the excerpts which I have above given will render this equally clear to my readers.

[1] *E.g.*, the localisation of appetite for food in the occipital lobes: 'Functions of the Brain,' 1st edition, 1876, p. 193.

poral gyri, Dr. Ferrier proceeds to invoke the evidence of two experimental observers who (as probably they themselves, in common with all the rest of the world, believed) have arrived at results which are diametrically opposed to this conclusion. It is certainly astonishing to hear the names of Munk and Luciani called as witnesses in favour of Dr. Ferrier's views on this point. Munk, in dogs, extends the area for perception of auditory impressions certainly over the whole of the temporal lobe—he distinctly states that he was not able to obtain similar evidence in monkeys; and Luciani did not succeed in obtaining permanent deafness with complete destruction of the cortex of the temporal lobe, even including the *cornu ammonis.* When, therefore, I stated that the supposed *localisation* (using the word in the sense above defined, which appears to me to be the only legitimate use of it) of the auditory perceptive faculties in the temporal lobe *in monkeys* (and *à fortiori* in the superior gyrus) has no experimental evidence in its favour, I merely recorded a demonstrable fact, and one which is still more patent, since the opportune publication of the notes on the monkey which was described as "manifestly totally deaf," but which nevertheless "evidently reacted to sounds."

Before leaving this subject of the superior temporal gyrus, it is necessary that I should refer—as briefly as may be—to Dr. Ferrier's statement, that I have charged him with misrepresenting me.

The statement is based upon the following note in the joint paper by Mr. Horsley and myself,[1] which is referred to by Dr. Ferrier, and which I think it will be well to give in full: "Dr. Ferrier is mistaken in the statement ('Functions of the Brain,' 2nd edition, pp. 310, 311), that we have been able to corroborate his observations upon the localisation of this (auditory) function in the superior temporo-sphenoidal gyrus in monkeys. We have so far neither obtained any distinct corroboration nor refutation of them, but regard the question as still open."[2] And in the text (same page) we say, speaking of the temporal lobe, "We have not as yet systematically pursued the question of the cortical localisation of auditory sensations, although it is right to state, that such incidental observations as we were able to make upon this function have almost uniformly proved negative. We have not as the result of any bilateral operation been able to convince ourselves that deafness has been established in a single case." And in a note to this we remark, "It is right to add that we were not specially intending to investigate this point (the localisation of auditory perceptions), and that the lesions of this lobe which we record were performed with another object, viz. to arrive at the hippocampal region. But, as we have been careful to note all the symptoms that we could observe, they are not without their value on this point."

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[1] 'Phil. Trans.,' 1888, B. p. 20.

[2] I had myself previously made a similar statement in a review of the 2nd edition of the 'Functions of the Brain' in 'Nature,' 1887, p. 465, in these

There is here no charge of misrepresentation; we merely remark that Dr. Ferrier has mistaken our, mostly verbal, communications, which were made to him during the progress of our experiments, and which were purely private and friendly and not intended for publication. Dr. Ferrier does not hesitate to seize upon a casual expression in a hurried note of invitation, in order to vindicate his position. In such a note, which was written at an early stage in the course of our joint work to ask him to come and inspect with us certain monkeys in which we had, as we thought, removed the whole temporal lobe of one side, including the hippocampus (which was the part we were at the time really trying to get at the function of), I mention as a reason for the invitation that we have been unable to detect any paresis of sensation (which, according to Dr. Ferrier's previous experiments, should have supervened), and I add in parenthesis "except auditory." Now this addition was made because it so happened that we were not certain with regard to

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words: "Dr. Ferrier is mistaken in supposing that the results of the experiments of Mr. Horsley and myself confirm his conclusions regarding the localisation of the auditory centre in the superior temporo-sphenoidal gyrus. The error seems to have arisen from the misunderstanding of a verbal communication. What we did find in one or two cases was that the whole of the temporo-sphenoidal lobe, exclusive of the superior gyrus, might be removed on both sides without loss of hearing,—not the converse, that hearing was abolished on destroying only the superior gyri on both sides. Indeed, we did not in any single instance perform this last experiment" one of the monkeys which we wanted Dr. Ferrier to test for hemianæsthesia, whether it was slightly deaf on the side opposite to the lesion.[1] The words "except auditory" were added to include this case and for the sake of being exact, certainly not for the sake of informing Dr. Ferrier that in all the monkeys from which we had removed the temporal lobe we had obtained deafness. If we informed Dr. Ferrier that this animal appeared to us slightly deaf when the ear of the same side was stopped, we must also have informed him that all the others, with equally extensive temporal lesions, showed no signs of deafness. But the question of audition was not raised at that time. We had not intended to deal with the localisation of auditory perceptions, and believed with most other people in this country in Dr. Ferrier's "auditory centre," as evinced by his "totally deaf" monkey. If in all the other animals with temporal lesions we failed to get any sign of deficiency of auditory perceptions, we were quite inclined to ascribe it to an incomplete removal, and at any rate we soon came to the conclusion, that there was nothing to be made out from monkeys in this direction by unilateral lesion, and left the question open for future investigation.

But I will assume for a moment, for the sake of the argument, that these two words "except auditory" (the occurrence of which I have above explained) might have been taken by Dr. Ferrier to mean, that we had found in all the several monkeys referred to in my letter (in which be it remembered the exact condition of the brain was not at the time known [2]) a deficiency of auditory perceptions.

In my letter I specify *the whole of the temporo-sphenoidal lobe*, including the hippocampus, as having been removed in the monkeys Dr. Ferrier is invited to come and see; how would this justify him in the statement: "My results have been *entirely confirmed* by Horsley and Schäfer, who have informed me that their experiments have shown, that it is *only when the superior temporo-sphenoidal convolution is destroyed that hearing is impaired or abolished,* and that no such effect is produced by entire removal of the rest of the temporo-sphenoidal lobe"?[1] It requires very clever casuistry to twist any sort of correspondence out of the two statements. Dr. Ferrier has not hesitated to charge me with want of ingenuousness,[2] but it seems to me that the accusation comes with ill effect from that quarter.

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[1] It was the only one in all our series of experiments that Mr. Horsley and myself had any doubt about at all. Our notes with respect to this monkey run thus ('Phil. Trans.,' vol. 179, 1888, B. p. 37): "30. *Lesion.*—Ablation of right temporo-sphenoidal lobe and hippocampus major. *Result.*—As regards sensibility, we could detect no difference on the two sides. As regards hearing, when the ear on the same side as the lesion is stopped, the animal seems unable to hear slight sounds." We have figured the lesion (Plate 5, Fig. 30), which is very considerable, extending even beyond the limits of the temporal lobe.

[2] Cf. Ferrier, op. cit. ('BRAIN,' April 1888, p. 9), who now strongly deprecates the "premature publication of conclusions while the evidence was incomplete, seeing that the animals were still alive and the actual lesions undetermined."

Our critic next comes to the consideration of the short paragraph in which I have dealt with the evidence afforded by our experiments on the functions of taste and smell, which were assumed by Dr. Ferrier, on what even he regards as insufficient evidence, to be *localised* in the antero-inferior extremity of the temporal lobe. After apologising for the weakness of the case, he concludes that, "inasmuch as Professor Schäfer, while unable to confirm, is also *unable to adduce any facts opposed to the positive results* obtained by (Munk, Luciani, and)[3] myself, it does not seem necessary to discuss the question at greater length." While agreeing with the last remark, so far as regards the mode of discussion of these questions which Dr. Ferrier has introduced, I may be pardoned for taking exception to the first, which I have italicised. The monkey whose brain is shown photographed in Figs. 1 *a* and 1 *b*, and in section in Figs. 1 *c* to 1 *g*, showed, after the first condition of idiocy[4] induced by the operation had passed off, complete possession of the faculties of both taste and smell. He was exhibited by us at a large meeting of the Neurological Society at which Dr. Ferrier was himself present; and the disgust which he manifested on biting a raisin in which quinine had been artfully concealed, and the care with which he afterwards smelt at every other portion of food which was offered to him, were sufficiently convincing to the members present that he both had and knew how to make use of these senses.

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[1] 'Functions of the Brain,' 2nd edition, pp. 310–311. The italics are mine. The statement in the latter part of the paragraph I freely admit.

[2] Op. cit., p. 12.

[3] I have ventured to put Munk and Luciani in parenthesis, because so far as I have been able to gather from their writings, their observations (made moreover chiefly, if not entirely, upon dogs) do not at all correspond with the results obtained by Dr. Ferrier.

[4] Cf. my paper in 'BRAIN,' January 1888, p. 375.

A single well-attested "negative instance" like this outweighs any number of positive instances. If an animal in which both temporal lobes have been destroyed so completely as is shown in these photographs still manifests to all the world that it continues to hear, to smell, and to taste, it is very improbable that any one of these three faculties is *localised* exclusively in this lobe.[1]

Lastly, with regard to the visual area of the cortex. Dr. Ferrier will have it that "the chief point at issue between him and myself is the anterior boundary of the visual zone. While he (Schäfer) considers it as being sharply bounded by the parieto-occipital fissure, I (Ferrier) maintain that it embraces also the angular gyrus."

This I cannot allow to be the chief point at issue, nor even that it is a point at issue at all. The chief points at issue are, (1) the connection of the angular gyrus with central vision of the opposite eye; (2) the relative importance of the occipital lobe. According to Dr. Ferrier's experiments, decorticisation of the angular gyrus of one side produces blindness of the opposite eye (amblyopia); of both sides produces blindness of both eyes. According to our experiments, decorticisation of the angular gyrus of one or both sides is not necessarily followed by any visual defects perceptible to our means of investigation in animals; but complete eradication of the gyrus produces hemiopia (not amblyopia), which is temporary only. According to Dr. Ferrier's experiments, destruction of one or both occipital lobes alone, produces no appreciable effect whatever on vision. According to our experiments—which are merely confirmatory of those of Munk—removal of one occipital lobe only, without the angular gyrus, produces permanent hemiopia; of both occipital lobes, blindness of both eyes, which is also permanent and, so far as we were able to judge, complete. Our experimental results therefore differ *toto cœlo.*

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[1] Not that these senses have nothing whatever to do with the lobe. This I have never asserted. I have, on the contrary, confined myself to the demonstrable statement, that the strict localisation of these faculties in the temporal lobe in monkeys has no experimental evidence in its favour. Whether with the lesions we have established there has been any permanent impairment of these senses could not by us, in our monkeys, be determined. All that we have ventured to state is that we have not been able to get evidence of such impairment. As for experiments upon dogs, it may be remarked that their results are not readily transferable to monkeys and man, owing to the extremely different configuration of the hemisphere, and that those who have experimented upon the sensory functions in these animals are by no means in agreement regarding the exact localisations of those functions. Indeed, Luciani—who, curious to relate, is again adduced by Dr. Ferrier as a witness for his case—extends the spheres for these special sense perceptions over areas of the cerebral cortex, so large and overlapping one another to so great an extent that they cannot in strictness be spoken of as "localisations" at all. (See 'BRAIN,' Vol. VII., figure opposite p. 160.)

In spite, however, of the experiments on total removal of the occipital lobe, and of this alone, which have produced in both Munk's hands and in our own permanent hemiopia or blindness, according as the lesion was on one or both sides, I have been careful to point out the possibility of the angular gyrus forming a part of the cortical visual area (loc. cit. p. 372), although it must be very subordinate to the occipital lobe. In this way the fact may be accounted for, that with eradication of the gyrus visual disturbance of a hemiopic nature is produced. At the same time, as will be seen from the photographic print (Fig. 4), which exhibits a longitudinal vertical section through the hemisphere, such eradication could not fail to affect the fibres of the corona radiata, which are passing to the occipital lobe; and the chief part of the hemiopia, which moreover is not permanent, might be accounted for by the mechanical insult to which these fibres are subjected by so deep a lesion. This is on the whole the more probable explanation, but there may nevertheless be left permanently a blindness of a small part of the visual field which it may be impossible to detect in monkeys. I at present have in my laboratory a monkey from which I have eradicated, as I believe, both *gyri angulares.* The operation was performed six months ago. For the first few days the animal appeared totally blind, but vision gradually returned, and before long was quite acute for distant objects. But for a long time, some three or four weeks, the animal failed to see objects (raisins, &c.) which were held just below or on one side of its eyes, and even now there appears to be complete absence of vision in the antero-superior and lateral portions of the retinæ. It is, however, difficult to prove this, because the creature has acquired the habit of rapidly directing his head and eyes, so as to bring the images of objects on the central parts of the retinæ. At an earlier stage it was very striking, for if a raisin were noticed at some little distance off, the animal would evidently see and run right up to it, but then often fail to find it, presumably because its image now fell upon the uppermost (blind) part of the retinæ. The early and temporary total blindness in this animal I should be inclined to explain by the mechanical insult of the thin part of the corona radiata which passes under the gyrus angularis to the occipital lobe (see Fig. 4), due to the eradication of the gyrus, and perhaps to pressure from the blood clot which occupies the place of the removed gyrus; the more permanent partial blindness (of the antero-superior and lateral parts of the retinæ) to the removal of the angular gyrus, and to some concomitant injury to the (corona radiata of the) lateral part of the occipital lobe, which, as I have elsewhere and by other methods shown,[1] are probably related to those parts of the retinæ. Dr. Ferrier, on the other hand, explains such blindness by supposing, that destruction of the gyri angulares produces loss of central vision. In that case it is difficult to account for the excellent central vision which the animal undoubtedly now possesses, unless the doctrine of cerebral localisation for vision is altogether to be surrendered, and we are to assume that other parts of the cerebral cortex are able rapidly to take up and to carry on the functions of the removed parts in addition to those functions which they already possessed.[2]

There remains one more criticism to be answered—that, namely, which alleges that in the monkeys in which we have produced permanent hemiopia or blindness after removal of the occipital lobe, we have extended the lesion to the under surface of the temporal lobe, and have thereby severed "the optic radiations from the corpora geniculata and primary optic ganglia." Optic radiations proceeding whither? Does Dr. Ferrier mean to imply, that the optic radiations to the gyrus angularis, "from the corpora geniculata and primary optic ganglia," run along the under surface of the temporal lobe to reach that gyrus? Is he not aware that the lateral ventricle and at least one deep horizontal fissure come between the region he speaks of and these "optic radiations"? And such "optic radiations" as pass into the occipital region, are they not tending towards the very parts which have been removed? A glance at any longitudinal section (such as that which is photographed in Fig. 4) will at once demonstrate the untenableness of the suggestion, that optic radiations proceeding to the angular gyrus can be at all involved in lesions of the occipito-temporal region.

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[1] 'BRAIN,' April 1888.

[2] Some thoughtful remarks, bearing relation to this point, will be found in the review by Mr. Lewes which has been already referred to.

Dr. Ferrier frequently refers to experiments of his own, and also to others by Mr. Horsley and myself, in which we obtained from circumscribed lesions of the occipital and occipito-temporal regions *temporary* hemiopic effects, and explains them all by supposing that our lesions invaded these "optic radiations." He says, "It is evident that the hemiopia observed in these experiments does not indicate the position of the visual centres. For from these facts alone we should have as much reason for placing the visual centre in the temporal as in the occipital lobe." Dr. Ferrier uses this argument as a *reductio ad absurdum*, but it is nevertheless very much to the point. There is, I think, no doubt whatever, both from these experiments in which hemiopia is produced from lesions of the occipito-temporal regions and from the results of excitation of the same region (which produces movements of the eyes upwards and to the opposite side[1]), that the visual area of the cortex *does* extend a certain distance forward beyond what is anatomically regarded as the anterior limit of the occipital lobe, especially on the under-surface. The hemiopia seems generally complete at first, but gradually passes off until the animal's vision appears fully restored. It is difficult, however, to say if any partial loss of vision remains. I believe the complete hemiopia to be due, in these cases of partial removal within the visual area, to the vascular and mechanical disturbance of the posterior region of the brain which is produced by the operations. This would sufficiently account for its temporary character. That a certain corresponding part of the retinal field remains permanently blind, I have very little doubt, but it is impossible to determine this in monkeys. At least, this seems to me a more reasonable explanation of the hemiopia than the assumption, that all such recorded lesions have extended to the "optic radiations." Whatever may have been the case in Dr. Ferrier's own investigations, this has certainly not happened in the experiments in which I have been engaged in conjunction either with Mr. Horsley or with Dr. Brown, as longitudinal sections through the hemispheres have abundantly shown.

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[1] Cf. the paper in the last number of 'BRAIN' on "Electrical Excitation of the Visual Area in the Monkey."

I share to the full the regret which Dr. Ferrier feels for the attitude I have taken up on this question, but not for my own sake nor for that of the truth. It is hardly necessary to say, that the only object I have had in view in making these experiments upon the brain has been to arrive at a more accurate knowledge regarding the functions of its parts. Few men have done so much towards the elucidation of the cerebral functions as my present opponent; and if we are not in entire accord with regard to some points, it is nevertheless to be borne in mind that the number of those upon which we are in complete agreement, and as to which I have had little to do but to confirm Dr. Ferrier's observations, is far greater. With respect to the points at issue I have merely endeavoured to state, as clearly as I could, the experimental facts which I have arrived at (conjointly with my fellow-workers), and at the same time to discuss their bearing upon the views which have been most widely accepted in this country regarding the cerebral functions. That we have finally settled the questions involved, I should be the last to affirm: I am content to have re-opened them. To time and to future observations, experimental and clinical, their decision may safely be left.

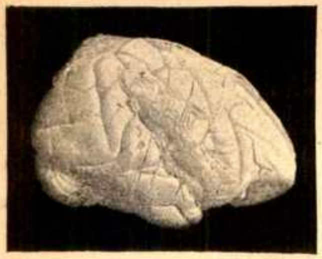

FIG. 2 *a*.

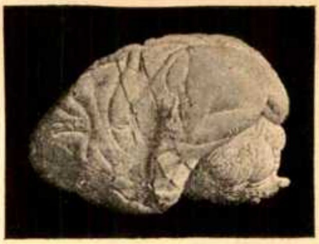

FIG. 2 *b*.

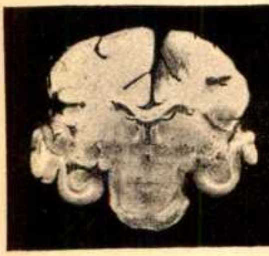

FIG. 2 *c*.

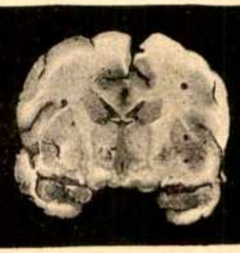

FIG. 2 *d*.

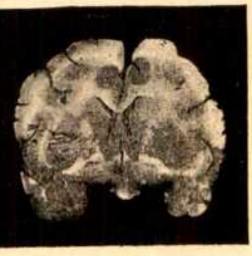

FIG. 2 *e*.

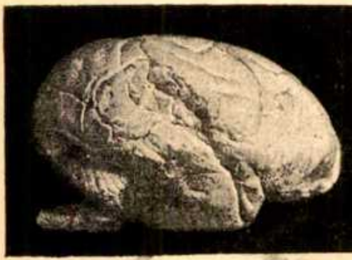

FIG. 3 *a* (reproduced from Ferrier and Yeo).

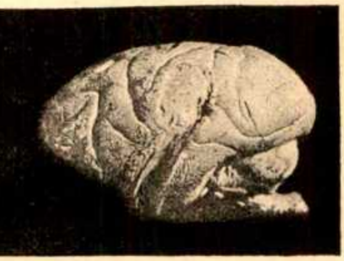

FIG. 3 *b*.

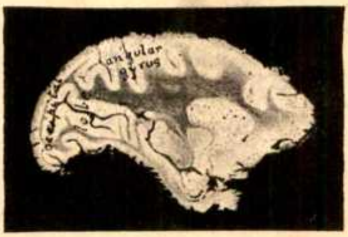

FIG. 4.

SCHÄFER: 'On the Temporal Lobes.'

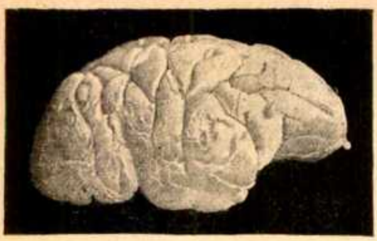

FIG. 1 *a*.

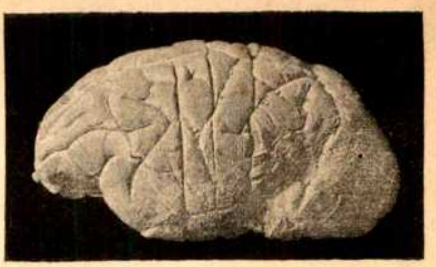

FIG. 1 *b*.

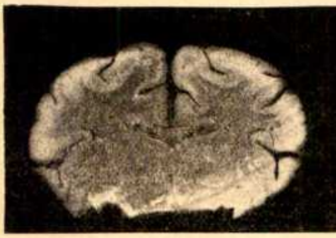

FIG. 1 *c*.

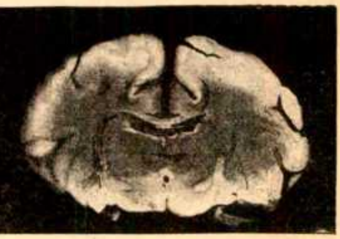

FIG. 1 *d*.

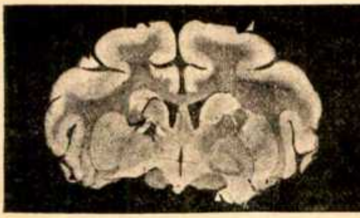

FIG. 1 *e*.

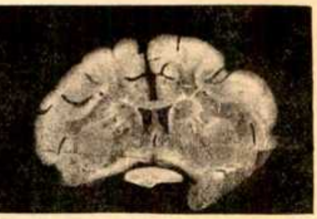

FIG. 1 *f*.

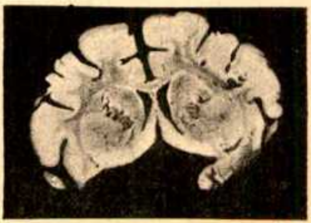

FIG. 1 *g*.

SCHÄFER: 'On the Temporal Lobes.'

At the request of Dr. Lauder Brunton,[1] I append a table, drawn up by himself, showing the methods he employed and the results he obtained in investigating, at the request of the Neurological Society, the monkeys which were submitted to the Society at its meeting on March 26, 1887. I should mention that Dr. Brunton at his own request was not informed of the nature of the lesion in the monkeys submitted to him, and, except on the last two occasions, other monkeys which had either not been operated upon at all, or which had had lesions established elsewhere than in the temporal lobe, were also handed over to him for investigation. The other members of the Committee who sent in reports (Dr. Bastian and Dr. Waller) content themselves with stating that, after carefully examining the monkeys submitted to them (F. and O.), they were satisfied that they could hear distinctly.

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[1] Who writes to me, "I see my notes of the reactions given by your monkeys have not appeared in 'BRAIN' yet. I should like them to do so, as I think it is important to have all the data before one in a case like the present, more especially as I see that my name appears in Ferrier's notes regarding another monkey, and people might imagine that I had had more experience in coming to a conclusion regarding the senses of monkeys than I actually have."

DESCRIPTION OF PLATES.

FIGS. 1 *a* and 1 *b*.—Photographs of brain of monkey F. (No. 6 of the cases recorded by Dr. Sanger Brown and myself in 'Phil. Trans.' 1888), from right and left side respectively.

FIGS. 1 *c* to 1 *g*.—Vertical sections through the above brain at successive levels from behind forwards. 1 *c* is taken through the level of the posterior extremity of the superior temporal gyrus; 1 *g*, through the tip of the temporal lobe. Only shreds, mostly detached, of the grey matter of the superior gyrus remain. The sections were made from collodion, so that these detached shreds have remained *in situ*.

FIGS. 2 *a* and 2 *b*.—Photographs of brain of monkey O, (No. 12 of the cases recorded by Dr. Brown and myself), from right and left sides respectively. The Sylvian and parallel fissures are blended into a broad gap, within which the Island of Reil comes to the surface—the superior temporal gyrus having been completely removed.

FIGS. 2 *c*, 2 *d* and 2 *e*.—Vertical sections through the above brain at successive levels from behind forwards. 2 *c* is taken through the level of the posterior extremity of the superior temporal gyrus: the injury has here extended to the Island of Reil. 2 *d* is taken through the level of the middle of the superior temporal, of which no fragment remains. All the other sections through the lesion show the same condition as fig. 2 *d* except those through the extreme tip of the lobe, where, for a length of about two millimeters, a shred of the grey matter (with its white centre destroyed) remains upon one side. This is shown in Fig. 2 *e*, on the right side of the section.

FIGS. 3 *a* and 3 *b*.—Photographs of the brain of the monkey (No. 13) exhibited to the Intern. Med. Congress in 1881 by Professors Ferrier and Yeo (reproduced from their paper in the Phil. Trans. 1884). The superior temporal gyrus is by no means so completely destroyed as in the other two brains.

FIG. 4.—Vertical sagittal section through one of the hemispheres of a monkey's brain, showing the relations of the angular gyrus to the corona radiata of the occipital lobe.

OBSERVATIONS ON MONKEYS BY DR. LAUDER BRUNTON.

As it seemed to be important to avoid any preconceived ideas which might render my observations fallacious, I remained in total ignorance of the nature of the lesion which had been inflicted upon the monkey until long after all the observations were finished.

Date.	Indication of Monkey.	Lesion inflicted. (This is subject to criticism.)	Tests supplied to determine the existence or absence of Hearing.	Results obtained.	Opinion formed.
May 9th, 1887.	Tame one (F.) ..	Very extensive double temporal, including whole of both superior gyri.	The door handle was shaken while the animal was looking steadily at me.	It did not look round the first time, but looked round when handle shaken more.	That the animal could hear.
"	Old Jew (H.) ..	Left occipital lobe only.	Door-handle shaken	The animal paid no attention.	
"	Three others ..	Had not been subjected to any operation.	Door-handle shaken. All sitting at top of cage.	No definite result	Doubtful whether they could hear or not.
"	Big female Rhoesus (O.).	Both superior temporal gyri removed.	Keys were shaken behind head	Looked round to right when I expected it would have looked to left.	That the animal could hear, but it seems quite deaf on left side.[1]
			The same thing repeated ..	Ditto.	
			Snapped finger close behind its ear.	No movement.	
			Struck two keys together.. ..	No reaction.	
			Mr. Green squeaked behind and to left of it.	First time it turned to right.	
			On repeating it	No reaction.	
			Struck bell behind my back ..	It looked round to right.	

			Again.	Ditto, although scratching arm at time.	
„	Old Jew (H.)	Same as above	Striking bell with hammer	No reaction	That the animal might be deaf.
„	Big female Rhoesus sitting with left side to door (O.).	Same as above	Door was opened and shut	Looked each time, and to left	That the animal could hear.
			This repeated	No definite reaction.	
			Water splashed on the ground	Looked up to left; couldn't well look to right.	
May 10th.	Big female Rhoesus (O.).	Same as above	Striking bell behind my back	Looked up to right	That the animal could hear.
			Knocking strongly on box	Ditto.	
			Whistles sounded	Gave no indication of hearing.	
Nov. 10th, 1887.	Large female Rhoesus (O.).	Same as above	Striking with a whistle or heavy piece of iron on top or side of cage.	Sometimes, but not always, moved its head as if to seek the sound.	That the monkey certainly could hear, while at the same time I do not think it reacts to sounds so readily and completely as a normal animal.[2]
				Sometimes gave no indication of hearing.	
„	When in the cage moved constantly to and fro.	Same as above	Fingers snapped; door-handle rattled; knock given on the table; foot scraped along the floor (especially the last).	It looked round (when removed from the cage and placed on a table).	

The particulars in the third column and the footnotes have been added by Professor Schäfer.

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[1] Apparently because it looked round to the right; afterwards, however, on the same occasion, it looked each time to the left (see below). [E. A. S.]

[2] This conclusion appears to me to lack evidence, considering that the only normal monkeys which were similarly examined by Dr. Brunton gave no definite indications of hearing (see above). [E. A. S.]

THE OBJECTIVE CAUSE OF SENSATION.—
PART III. THE SENSE OF SMELL.

BY PROF. JOHN BERRY HAYCRAFT.

THE end-organs of the special senses are all built up on the same type. The history of their development from simple ectodermic cells suggests that similar agencies have been at work to produce them. Both sapid and odorous substances, and indeed all gaseous and liquid molecules, are now known to be in constant vibration, and this vibration is more or less characteristic of the substance examined.

The above considerations have led me, for the last five years, to teach that, in all probability, it will be possible to connect *quality* of taste and smell with the *kind* of vibrating stimulus, and that it will be possible to demonstrate, as has already been done in the case of sight and hearing, the truth of this general statement—that quality of sensation will depend (the sensorium being in a normal condition) upon the kind or character of the vibrating stimulus. There is nothing very new in this idea. Without seeking for its germs at an earlier period, we find it clearly enunciated by both Hobbes and Hartley; and in more recent times Mr. Herbert Spencer has lent the weight of his great authority in the same direction. But of experimental proof, without which we cannot rest content, nothing has been advanced. Casual allusions to the probable or possible relationship between the tastes or smells of bodies and their chemical natures, are sometimes though rarely found in the text-books of physiologists, but nothing more. While so much important work has been carried on during the last few years by Helmholtz, Preyer, Maxwell, Rayleigh, and others, in connection with both sound and sight, no one, until quite recently, has turned his attention to the investigation of either taste or smell.

In a most interesting and suggestive article in 'Nature' (June 22, 1882), Prof. Ramsay brought forward many facts tending to demonstrate the dependence of *smell* upon the vibratory motion of odorous particles. He drew attention to the fact, that many gases and vapours of low specific gravity—their molecules vibrating therefore with great rapidity—are perfectly odourless, and he saw in this an analogy to the rapid vibrations of the ultra-violet rays of the spectrum, and the rapid vibrations of an insect's wing, both incapable of producing any impression on the eye and ear. He also described classes of substances alike in chemical and physical properties, such as the alcohols or fatty acids, as having generic smells; the higher members of the groups producing sensations more powerful and characteristic than those of the lower ones. It will be my endeavour in the present paper to extend more fully this inquiry, and to demonstrate by experimental methods the fact that smell, like sight and hearing, depends for its production on the vibrations of the stimulating medium, the quality of the sensation depending, in all cases, upon the kind of vibration which produces it.

In a paper read before the British Association in 1885, and printed subsequently in the 'Proceedings of the Royal Society of Edinburgh' (1886), I was able clearly to demonstrate these points for the sense of taste. That paper and the present one will be found to run on exactly parallel lines; one is almost a recapitulation of the other, for what is true of taste is also true of smell. In order to avoid unnecessary recapitulation, I have touched lightly on many questions more fully discussed in the other paper, which should therefore be consulted.

An investigation into the odorous properties of substances is to a certain extent limited, as many of them are without smell, especially those found in the inorganic world. In a description of odours one is met with this difficulty, that there is no nomenclature familiar to every one. Hundreds of terms expressing the well-known colours of familiar objects, enable one to describe by a single term almost any tint and shade. We have cardinal, rose, magenta, maroon, carmine, crimson scarlet, and a dozen other shades of red alone, and all of these can be expressed by words. The smells, however, and especially those of the chemist's museum, are so unfamiliar, and often so peculiar, that we are forced to speak of them simply as the odours of the substances which produce them, or to say that they are like, though seldom identical, with that of some other and better known substance.

No two observers quite agree in their descriptions of a given odour, and the information readily at hand in the text-books, but culled from a hundred sources, is therefore not reliable. I have, for this reason, availed myself freely of the kindness of Prof. Tilden and my friend Prof. Ramsay, who have placed at my disposal their private collections of chemical compounds. In almost every case the description of a smell given in this paper is derived from personal observation.

In the first place, let us study those few substances found among inorganic compounds which have distinct smells. It is well known that many substances, like arsenic, chlorine, sulphur, bromine, and their compounds, have characteristic odours. Can we associate the odours of these substances with any chemical or physical properties they may possess, and show that when similar odours are produced by two or more substances, then we have some similar chemical or physical property present at the same time?

In recent years a remarkable discovery of Newlands has opened up a fresh point of departure in the science of chemico-physics. His observations led him to formulate a law which he termed the law of octaves. Lothar Meyer, Mendelejeff, and Carnelley, extending his work, have shown that the "periodic-law," as it is now called, is one of vast application and importance. The nature of this periodic-law is now so well known, thanks to the many recent publications of Professor Carnelley, that it would be superfluous to attempt more than roughly to sketch out its main features. If we arrange the elements in the order of their atomic weights, beginning with that which has the lowest, and passing to that which has the highest, we shall find a periodic recurrence of property or function in the series. The first element is a monad, the second a dyad, the third a triad, and the fourth a tetrad. Then we find the fifth a triad, the sixth a dyad, and the seventh once more a monad. Then follows a second series of seven elements, showing the same variation in atomicity; this repeats itself right through the list of elements. This periodic recurrence of function is seen not only in the case of atomicity, but it may also be observed in the atomic volumes, the fusibility and the electrical and other properties of the elements. There is then a general resemblance in physical properties between the first, eighth, fifteenth, &c., and between the second, ninth, and sixteenth elements. Mendelejeff has arranged the elements in the convenient tabular form given on the opposite page, which indicates these and some other important facts.

Those elements which resemble one another, and which we can pick out by taking every eighth one from that other from which we elect to start, form what he calls a "group," and are arranged vertically. The sets of seven elements each, arranged horizontally, form twelve "series."

There is yet another point of importance. The elements of a "group," which are in an even "series," are especially related to one another; so in like manner elements in an old series of the same group are similarly allied. Thus Li, Na, K, Cu, Rb, Ag, Cs, Au, have all these properties in common; but in this group Na, Cu, Ag, Au are most alike, and Li, K, Rb, and Cs, in like manner, are most closely related.

In the paper to which I have already alluded, I was able to demonstrate the fact, that elements in the same group are capable of producing similar or related tastes. The power of producing a given taste is then a property which, like the ordinary physical qualities of the elements, follows the periodic law. As will now be shown, the same obtains for smell.

In studying the facts of the case, let us start with Group VI. We find here, in odd series, three well-known substances whose compounds have strong and characteristic odours. Sulphuretted, selenietted, and telluretted hydrogen have all a disagreeable odour, like that of rotten eggs. The compounds

TABLE OF NATURAL CLASSIFICATION OF ELEMENTS—*After Mendelejeff.*

Groups.	I.	II.	III.	IV.	V.	VI.	VII.	Group VIII.
Series.	Monads.	Dyads.	Triads.	Tetrads.	Triads or Pentads.	Dyads or Hexads.	Monads or Heptads.	
1.	H = 1	...	...	...	...	...	...	
2.	Li = 7	Be = 9	B = 11	C = 12	N = 14	O = 16	F = 19	
3.	Na = 23	Mg = 24	Al = 27	Si = 28	P = 31	S = 32	Cl = 35·5	
4.	K = 39	Ca = 40	Sc = 44	Ti = 48	V = 51	Cr = 53	Mn = 55	Fe=56. Co=59. Ni=59.
5.	Cu = 63	Zn = 65	Ga = 69	...	As = 75	Se = 79	Br = 80	Ru=104. Rh=104. Pd=106.
6.	Rb = 85	Sr = 87	Y = 89	Zr = 90	Nb = 94	Mo = 96	...	
7.	Ag = 108	Cd = 112	In = 113	Sn = 118	Sb = 120	Te = 125	I = 127	
8.	Cs = 133	Ba = 137	La = 139	Ce = 142	Di = 147	...	...	
9.	...	...	...	...	Er = 166	...	...	
10.	...	...	Yb = 173	...	Ta = 182	W = 184	...	Os=193. Ir=193. Pt=195.
11.	Au = 197	Hg = 200	Tl = 204	Pb = 207	Bi = 210	...	...	
12.	...	...	...	Th = 234	...	U = 240	...	

of the elements of this group with methyl and ethyl are disagreeable and alliaceous. In Group VII. chlorine, bromine, and iodine have very similar smells, and so have the acids they form with hydrogen, and their compounds with methyl, ethyl, ethylene, &c. Although similar in all cases, yet they are not the same. One can distinguish the odour of iodine from that of bromine or chlorine. It may be described as having more flavour, and not so chlorous. Bromine is like them both, having an odour intermediate between the two in quality. From a study of the above substances, this is seen to hold good in all cases, the odour uniformly changing, often to a slight degree, as we pass from the lowest to the highest member of a group. A very marked change is seen in the formyle compounds of Group VII. Chloroform has a fragrant and characteristic smell. So has bromoform, but it has something else in addition, which is recognised as being the odour of iodoform. Bromoform thus connects chloroform and iodoform, these latter substances being very unlike one another.

There may then be so little difference in the odours of compounds of the same group of elements that they may with difficulty be distinguished. On the other hand, the differences may be great, there being intermediate sensations produced by intermediate members of the group. One is forcibly reminded of the changes in sensation experienced in allowing the eye to traverse the spectrum from one end towards the other. In a drawn-out spectrum, only a part of which is visible, one passes, say, from orange into yellow, and these colours are recognised as being different, and at the same time alike. In a shorter spectrum the eye may pass from the yellow into the red. The two sensations are quite different, but the orange is seen to connect them. The importance of the above statements—and they may be verified in the case of the few odorous compounds in Group V.—will become apparent when they are placed in juxtaposition with identical facts mentioned in the previous paper on taste, and some recent observations of Professor Carnelley.

In those groups of elements whose compounds are sapid, we find that the same change in taste sensation is apparent as we pass from lower to higher members of the group. Let us take as an example Group VII., which furnishes us both with sapid and odorous bodies.

GROUP VII.

Element.	Potassium Compound.	Formyle Compound.
F	Salt and *saline.*	...
Cl	Salt, saline, *bitter.*	Characteristic odour of chloroform.
Mn	——	——
Br	Salt, saline, and *bitter.*	Intermediate odour.
I	Saline, bitter.	Characteristic odour of iodoform.

Potassium fluoride is salt—like common salt in taste. In addition it is slightly saline—like nitre. Chloride of potassium has a suspicion of a bitter taste as well, and so has potassium bromide. Potassium iodide has lost the taste of common salt, being a saline, and bitter.

So far, then, we have seen that the power of producing taste is a property or function of elements. Our knowledge of matter is derived from its power of producing sensation within us. In the case of sight and hearing we have associated quality in sensation with the kind or character of the vibrating stimulus. If it can be shown that elements belonging to the same "group" are capable of vibrating in a way which is similar or related to one another, then we have grounds upon which to draw an analogy between smell, taste, sight, and hearing.

On account of our incomplete knowledge of the ultra-red and ultra-violet regions of the spectrum, a final answer to this question cannot perhaps be given. Only rough indications are to hand, but these point in the same direction.

The chlorides of the alkaline earths have spectra which are nearly related. The spectra of Group I. are not dissimilar, especially potassium and rubidium, with the five groups of lines. Then, again, the chlorides, bromides, and iodides of calcium and barium are similar, *the lines shifting towards the red end of the spectrum* in a way which is nearly proportional to the increase of atomic weight.

The colour of a substance is an index to the pitch of the vibrations of its molecules. In a paper on the colour of chemical compounds recently published in the 'Philosophical Magazine' (July 1864), Professor Carnelley demonstrated the existence of a relationship between the salts of the same group of elements in respect to colour. The salts, say the chlorides, of a group of metals may not be of the same colour, but we find that, in passing to higher members of the group from the lower ones, a uniform *change* in colour is to be observed. This change is produced by a gradual shifting of the absorption towards the red end of the spectrum, the molecules vibrating more and more slowly with increase of atomic weight. This can be illustrated by the following diagram, taken from his paper :—

Metal.	Cl	I	Metal.	CrO_4	AsO_4
Na	White	White	Mg	Lemon yellow	White
Cu	White	Cream	Zn	Yellow	White
Ag	White	Light yellow	Cd	Orange yellow	White
Au	Yellow white	Golden yellow	Hg	Red	Yellow

It is probable, then, that metals of the same group vibrate in a similar way. This vibration we know is complex, consisting of many wave-lengths of different pitch. When a metal vibrates, any one of its vibrations falls within the scale of the visible spectrum, we shall find the corresponding wave of another member of the same group in the neighbourhood. If a higher member of the group, it will absorb the light nearer the red end; if a lower, nearer the blue end of the spectrum. Together with this alteration of pitch, we have corresponding alterations in the sensations produced, whether they be of sight, taste, or smell. Whatever reason we have for associating quality of colour with the pitch of vibration, we shall likewise have for associating quality of smell and taste with the same physical cause.[1]

Amongst organic substances many are so closely allied that they fall into distinct classes or groups. Thus we have the fatty acids, alcohols, &c. If these be arranged in homologous series, commencing with that which has the lowest, and passing to that which has the highest molecular weight, a uniform change will be observed in many physical properties on ascending the series. Thus the lower ones may be gaseous, the middle ones liquid, and the higher ones solid.

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[1] If a curve be constructed in which the ordinates represent the atomic weights of the positive elements, and the abscissæ a chromatic scale arising from blue, green, &c., to black, we shall obtain a curve indicating that the colours of the compounds are a periodic function of the elements arranged in atomic series. This is well seen in the case of the normal iodides (Carnelley).

Professor Ramsay examined several of these groups, and came to the following conclusions:—In the first case, that the smell of a group was generic; and, in the second case, that the smell became more distinct, and gained in flavour in ascending from the lower to the higher members. My observations are not quite in accordance with the first statement, for I do not believe any uninitiated person would find any resemblance, say, between the odours of ethyl alcohol and octyl alcohol, or of acetic and valeric acids, which would prompt him in any way to class them together. I find that in ascending the organic series, as in ascending one of Mendelejeff's groups, the odour changes. This change may be slight, so that it may be said with truth that there is a generic smell belonging to the series; or, more frequently, the change is so great that it is only by a study of intermediate members that any continuity of sensation can be made out.

These statements may be verified by a study of the following tables. They are not as complete as I could have wished, owing to my inability to obtain some of the rarer acids and alcohols.

Monatomic Alcohols.

$CH_3.OH$	Methyl alcohol		=	Faint alcoholic odour.
$C_2H_5.OH$	Ethyl	„	=	Alcoholic odour.
$C_3H_7.OH$	Propyl	„	=	Alcoholic odour with flavour.
$C_4H_9.OH$	Isobutyl	„	=	Flavour becomes more marked, and the alcoholic odour less and less.
$C_5A_{11}.OH$	Amyl	„	=	
$C_8H_{17}.OH$	Octyl	„	=	

The term "flavour" expresses very badly what is meant. It is only possible by experiment to become acquainted with the nature of a smell.

Among the fatty acids another flavour equally characteristic gradually supersedes the acetic odour of the first two members of the group.

Fatty Acids.

$CHO.OH$	Formic acid	=	Acetic odour.
$C_2H_3O.OH$	Acetic „	=	Acetic odour.
$C_3H_5O.OH$	Propionic „	=	Acetic, together with a flavour.
$C_4H_7O.OH$	Butyric „	=	Slightly acetic, with a well-marked flavour.
$C_5H_9O.OH$	Valeric „	=	No longer acetic, the flavour alone is present.

In this case the smell has altogether changed in character. The same holds good with the following series.

Acetates.

$C_2H_5.C_2H_3O_2$	Ethyl acetate	=	Acetic and ethereal smell.
$C_3H_7.C_2H_3O_2$	Propyl „	=	Acetic ethereal, with a flavour.
$C_4H_9.C_2H_3O_2$	Butyl „	=	Slightly acetic, with a pine-apple flavour.
$C_5H_{11}.C_2H_3O_2$	Amyl „	=	Pine-apple flavour and not acetic.

If we pass to quite another group, the hydrocarbons, and starting with benzene, replace first one, then two, and finally three atoms, with methyl, the ethereal aromatic odour will be found progressively to change in a manner which it is impossible to describe, but which can readily be demonstrated.

Hydrocarbons.

C_6H_6	Benzene	Have a progressing and aromatic and ethereal odour.
$CH_3.C_6H_5$	Methyl benzene	
$2(CH_3).C_6H_4$	Dimethyl benzene	
$3(CH_3).C_6H_3$	Trimethyl benzene	

The way in which the sensation changes, analogous to that observed in studying Mendelejeff's groups, can in a similar way be explained on the vibration hypothesis.

I am not aware of any odorous organic series possessing at the same time colour, although some of them have very weak absorption bands. From a study of these latter, and from inferences drawn from a study of other coloured series, it is possible to obtain an insight into the state of vibrational activity of the substances in the tables above. Dr. W. J. Russell has investigated the absorption bands of ammonia, alcohol, &c. These substances absorb light, but to so slight an extent that long columns of the liquids have to be examined before the bands are distinctly seen. Under these conditions, ammonia gives several distinct and characteristic bands. If now an atom of hydrogen of the ammonia be replaced by methyl, the ammonia bands are still visible, but they are shifted somewhat towards the red end of the spectrum. Replacing the hydrogen by the larger molecule of ethyl, the bands are seen to pass still nearer to the end of the spectrum.

NH_3	Ammonia	Produce bands which shift to red end of spectrum in ascending the series.
CH_3NH_2	Methylamine	
$C_2H_5NH_2$	Ethylamine	

In the same way, we find that common alcohol possesses absorption bands, seen also in higher members of the group, but shifting towards the red end of the spectrum in ascending the group.

$C_2H_5.OH$ Alcohol	Produce bands which shift to the red end of the spectrum in ascending the series.
$C_3H_7.OH$ Propyl alcohol	
$C_4H_9.OH$ Butyl alcohol	

In the case of coloured acids, such as chromic and picric acids, the salts too are coloured. If the bands of these acids be examined, and if they be then converted into salts, the absorption will shift towards the red end of the spectrum. It seems that the molecule, having a certain vibrational character depending upon its structure, is weighted by the added metal, the vibrations of which do not probably appear at all in the visible spectrum, and, in consequence, its pitch is lowered. If an odorous substance like acetic acid be combined with another odorous substance, it is generally possible to detect the two intermingled sensations in the compound. Ethyl acetate is ethereal and acetous at the same time. Allyl sulphide is like allyl alcohol, and has the odour of a sulphur compound as well. In this case, it is probable that those vibrations in each substance which produce smell are not so much lowered in pitch by the new substance with which they are combined as to change the character of the sensations they are each capable of producing. The ammonia vibrations are shifted towards the red end of the spectrum in methylamine, but not enough to produce another sensation. In other compounds of two odorous substances it may not be possible to distinguish the original odours, and for the reason that the pitch has shifted, as we have seen it often does, so as to produce quite a different sensation.

It may be urged as an objection to some of these conclusions that the same odours are often produced by substances, chemically speaking, quite unlike each other. Thus benzoic aldehyde smells very much like nitrobenzene. In the case of taste, too, there are many examples of totally dissimilar bodies having indistinguishable acid or sweet tastes. In answer to this objection one has only to remember, that there are instances, equally numerous, of very different substances which produce the same colour sensations. One may produce exactly the same tint with either a chromate, a picrate, or an aniline dye. It would be strange, indeed, if among the complex vibrations of a compound, or even of an element, some tones were not of the same pitch, as some of the vibrations of substances quite dissimilar in general properties. When these tones fall within the scale of the visible spectrum, the scale of taste, or smell sensations, we have, according to the vibration theory, a similar sensation produced.

In this paper I have endeavoured to avoid all questions which are matters of speculation. I have dealt only with already ascertained facts, or those which can readily be verified. I do not attempt to offer any hypothesis to account for the action of vibrating matter on the olfactory end-organs. It may or may not be a mechanical or chemical action. This question is not raised. We know next to nothing as to how it is that ether vibrations stimulate the cones of the retina, still less can we guess at the action of vibrating atoms and molecules of ordinary matter on the sensitive end-organs of the nose. My aim has been to establish the fact that, just as we have reason to connect differences in colour sensations with differences in the vibration of the ether, so, in like manner we have reason to connect differences in smells with differences in the vibrations which call them into existence. This analogy is established upon the following grounds :—

(1) In passing from the lower to the higher members of one

of Mendelejeff's groups, such molecular vibrations as have been investigated tend to become lower in pitch. At the same time the colour, taste, and smell sensations alter in character when present.

(2) In passing from the lower to the higher members of an organic series, such as the alcohols, such molecular vibrations as have been investigated tend to become lower in pitch. When present, the colour, taste, and smell sensations alter in the manner I have described.

ON A PARTICULAR VARIETY OF EPILEPSY ("INTELLECTUAL AURA"), ONE CASE WITH SYMPTOMS OF ORGANIC BRAIN DISEASE.

BY J. HUGHLINGS-JACKSON, M.D., LL.D., F.R.S.

I HAVE notes of about fifty cases of the variety of Epilepsy I am about to speak of. I have seen very many patients with symptoms of local gross organic brain disease (optic neuritis, &c.); in many of the latter, as subsequent necropsies showed, there was intracranial tumour. But one of the cases (Case 1, p. 191) I am about to relate and remark on (I have referred to it briefly, 'Bowman Lecture,' "On Ophthalmology and Diseases of the Nervous System," 'Trans. Ophth. Soc.' vol. 6), is the only one I have seen in my own practice in which this variety of epilepsy was found associated with marked symptoms of local gross organic brain disease.[1] Although necropsy was forbidden, the case is of great clinical importance. The variety of epilepsy alluded to is one in which (1) the so-called "intellectual aura" (I call it "dreamy state") is a striking symptom. This is a very elaborate or "voluminous" mental state. One kind of it is "Reminiscence"; a feeling many people have had when apparently in good health (see p. 184, the case of Quærens and that of Dr. Ferrier's patient, Case 3). Along with this voluminous mental state, there is frequently a "crude sensation" ("warning") of (*a*) smell or (*b*) taste; (or, when there is no taste, there may be movements, chewing, tasting, spitting, *implying* (?) an epileptic discharge beginning in some part of the gustatory centres), or (*c*), the

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[1] Since this was written I have had a necropsy of a woman who had had paroxysms with the "dreamy state," and crude sensation "warnings" of smell. She had left hemiplegia and double optic neuritis. I can now only say that there was a tumour in the right temporo-sphenoidal lobe. My colleague, Dr. Beevor, who sent the patient to me, has kindly undertaken the examination of the specimen; on receiving his report I shall publish the case.

"epigastric" or some other "systemic" sensation. The wording of this statement implies, at any rate it is meant to imply, that the "dreamy state" sometimes occurs without any of the crude sensations mentioned, or movements supposed to imply discharges of gustatory elements, and that sometimes those crude sensations and movements occur without the "dreamy state"; this will be exemplified in cases shortly to be given for incidental illustration.

I have been struck by certain non-associations. In my experience vertigo, in the sense of external objects seeming to move to one side, rarely occurs with the "dreamy state." In this paper I have to state exceptions (see Case 2) to this. The other variety of vertigo, that is, the feeling of the patient himself turning, does not so rarely occur with the "dreamy state." Again, I have no account of crude sensations of sight (colour projections) associated with the "dreamy state," but I have notes of one case in which the patient, *at other times,* had migrainous paroxysms with visual projections. In cases of epilepsy beginning by colour projections, the much less elaborate mental state "seeing faces," is not uncommon. I have thought that crude sensations of hearing are not associated with the "dreamy state." Until recently I have known of no exception, but I shall have to relate one in a case, the notes of which are supplied to me by Dr. James Anderson. Auditory sensation-warnings are not rarely followed by "hearing voices" (really words as if spoken to the patient), a less elaborate state than the "dreamy state." I now return to the variety epilepsy with the "dreamy state."

There is not always *loss,* but there is, I believe, always, at least *defect,* of consciousness co-existing with the over-consciousness ("dreamy state"). *After* some paroxysms in which consciousness has been lost there are exceedingly complex and very purposive-seeming actions during continuing unconsciousness; in a few cases the actions appear to be in accord with the "dreamy state."

It will have been seen that I do not consider the "dreamy state" to be a "warning" ("aura"), that is to say not a phenomenon of the same order as the crude sensations of smell, &c. Hence my objection to the term "intellectual aura," and adoption of the less question-begging adjective "dreamy," one which is sometimes used by the patients. It is very important in this enquiry to distinguish mental states according to their degree of elaborateness—from crude, such as the crude sensation-warnings of smell, &c., to the vastly more elaborate, such as the "dreamy state"—in order that we may infer the physical condition proper to each. The crude sensations are properly called warnings; they occur during *epileptic* (sudden, excessive and rapid) discharges; the elaborate state I call "dreamy state" arises during but slightly raised activities (slightly increased discharges) of healthy nervous arrangements.

I have previously considered this variety of epilepsy, 'Med. Times and Gazette,' Dec. 2nd, 1876, and Feb. 1, and March 1, 1879; 'BRAIN,' July, 1880. These papers have attracted very little attention; they have, however, been referred to by Dr. Mercier; by Dr. Beevor, in his important article "On the Relation of the 'Aura' Giddiness to Epileptic Seizures," 'BRAIN,' January, 1884, p. 488; and by Dr. James Anderson (*vide infra*, p. 182). The following quotation is from a lecture I published, 'Med. Times and Gazette,' March 1, 1879, p. 224:—"I think it will be found that in many, I dare not say in most, cases the voluminous mental ['dreamy'] state occurs in patients who have at the onset of their seizures some 'digestive' sensation—smell, epigastric sensation, taste, or, in cases where there are movements implying excitations of centres for some such sensations, such movements as those of mastication."

Under the name "intellectual aura," the "dreamy state" has long been known to occur in epileptics. The case of Quærens (*vide infra*, p. 184) is, so far as I know, the first definite case of epilepsy with that phenomenon published in this country. Dr. Joseph Coats, 'Brit. Med. Journal,' Nov. 18, 1876, has recorded a very important case of an epileptic whose fits, with few exceptions, were preceded by giddiness and a "peculiar thought." "Sometimes the fit only consists of the aura [the thought], followed by a peculiar feeling in the abdomen which passes up to the head and back to the abdomen, when vomiting results."

Dr. James Anderson has recorded a case of this variety[1] of epilepsy in which, from symptoms, ocular and cerebral, detailed in his report, he correctly predicated tumour, and its position. This case has several important bearings, but for my present purpose it will suffice to say, that the patient's "dreamy state" was associated with a rough "bitter sensation" in his mouth. It is the only case published which I know of in which a necropsy has been had revealing any local morbid changes in a case of the variety of epilepsy mentioned. Dr. Anderson refers to a case, closely like that of his own patient, recorded by Mr. Nettleship, 'Trans. Ophth. Soc.' vol. iv. (Necropsy by Dr. Sharkey). In the report of that case, however, the "dreamy state" is not mentioned; there was a crude sensation warning in the patient's fits, "a sudden feeling of suffocation in the nose and mouth." I think it not impossible that the "dreamy state" was present in the slight seizures (the patient did not always lose consciousness). I doubt not that I have in former years disregarded this important symptom. I have suggested ('Bowman Lecture,' *op. cit.*) that ophthalmic surgeons who see very many cases of optic neuritis (that is, cases in most of which there is local gross organic intracranial disease, such as tumour) should minutely investigate any paroxysms, however slight and transient, their patients may have, especially when there is any kind of defect of smell or taste. Just as the most exact knowledge we have of the seats of "discharging lesions" in different epilepti*form* seizures is from cases of gross local organic brain disease, so no doubt our most exact knowledge of the seats of "discharging lesions" in epilep*tic* seizures will be obtained from cases of such kind of disease. Some preliminary remarks on *slight* epileptic fits are necessary. I mean fits commonly called attacks of epilepsy proper.[2]

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[1] "On Sensory Epilepsy. Case of Basal Cerebral Tumour, affecting the left Temporo-Sphenoidal Lobe, and giving rise to a Paroxysmal Taste-sensation and Dreamy State:' 'BRAIN,' Oct. 1886.

[2] Using the colourless word "fits" generically, I make three classes of fits (see 'BRAIN,' April, 1886): (1) Ponto-Bulbar; (2) Epileptiform; (3) Epileptic (Epilepsy proper of nosologists). As the name implies, (1) depends on discharges beginning in bulbar and pontal centres (laryngismus stridulus, certain uræmic fits (?) and asthma? and I imagine some fits *called* epileptic). I have published ('Brit. Med. Journ.', Nov 20, 1886) the case of a boy who had fits started by

The slighter paroxysms are, the more deserving are they of minute and precise investigation, both for the patient's sake and for scientific purposes; for the patient's sake since, unless we give most careful attention to the details of them, we shall sometimes altogether overlook epilepsy; for scientific purposes, because the analysis of slight seizures is more easy and fruitful than that of severe ones. It often happens that a patient has sometimes slight seizures of the variety of epilepsy under remark, and at other times severe seizures; and not rarely he has no "warning," in any sense of the term, of the latter. Obviously the clue to the seat of the "discharging lesion" is only given definitely by the "warning" (such as the crude sensations mentioned); so that of the patient's slight seizures we may learn much, of the severe ones without warning very little that is definite.

I urge strongly that the great thing as to the diagnosis of epilepsy is not the "quantity" of the symptoms, nor the severity of the fits, but paroxysmalness. Again, *loss* of consciousness is not essential for the diagnosis of epilepsy; there may be *defect* of consciousness only; and, as we have been saying, there may be "over-consciousness" ("dreamy state") co-existing with the defect of consciousness; with defect of consciousness as to present surroundings there may be a rise of consciousness as to some other and often quasi-former surroundings ("dreamy state"); the latter may attract exclusive attention, the co-existing defect of consciousness being ignored. The most seemingly trifling symptoms, when occurring paroxysmally, deserve careful analysis in proportion to their paroxysmalness; suddenly "coming over queer" for

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touching his head, a case analogous to fits artificially produced in guinea-pigs (Brown-Séquard), and due, I presume, to abnormal changes in the ponto-bulbar region. Class (2) is of fits depending on discharge beginning in some part of convolutions of the so-called "motor region." I imagine that (3) is owing to discharge beginning in some part of convolutions of the cerebrum other than those of the "motor region." Both (2) and (3) are to my mind "cortical," although that term is commonly given to (2) only. I think it most likely that migrainous paroxysms are "fits" which are the (chiefly) sensory analogues of (2) epileptiform seizures. I feel confident that (3), epilepsy proper, will have to be subdivided very considerably, and possibly some seizures we call epileptic will have to be classified apart. I hope the above classification will be useful provisionally.

a moment or two, may be a slight epileptic attack and the forerunner of severe attacks. Of course it is a very old story that veritable epileptic fits may be very slight indeed, and, often enough, so slight and transitory that bystanders do not notice them; but there are particular reasons for insisting on this point with regard to cases of the variety of epilepsy the subject of this paper. I particularly wish to remark that, in many of them, the slight seizures are so very slight, that the patient unfortunately disregards or underrates them until a severe fit comes and declares their evil significance. As bearing closely on this neglect, I here say that such slight seizures are not always disagreeable, but sometimes positively agreeable. I have heard patients say that they used to "encourage" the feeling, before they knew what it meant. The day I write this, a patient told me that he used to try to bring the feelings on when he first had the attacks; they are now disagreeable. The symptoms often seem to be so fanciful to the patients that they may reckon them for a time as mere oddities. Even when they have found out the bad meaning of their slight attacks, they are often seemingly unwilling to give any details of the "dreamy state." Dr. James Anderson's patient "showed some reluctance to talk about the scene." They and their friends do not seem to care for questions as to movements of chewing, smacking the lips, &c., thinking, probably, that such little things have no real bearing on a serious condition. I would go further and say, that some medical men seem to think questionings on the "dreamy state," enquiries about spitting, champing movements, &c., are unpractical. I now stay to illustrate some of the preceding remarks.

One of my patients (*vide infra*, Case 5), a medical man, had seizures of this variety of epilepsy in so slight degree at first, that he took no more notice of them than to make them a subject of joking (to use the words from the report he made of his own case, he "regarded the matter playfully, as of no practical importance"). He now has severe as well as slight fits. I refer also to the case of a medical man who reported it himself under the pseudonym Quærens ('Practitioner,' May 1874, p. 284). The title is, "A Prognostic and

Therapeutical Indication in Epilepsy." When he consulted me, Feb. 1880, he had had eighteen severe fits (loss of consciousness, convulsion, tongue biting), and had had "many hundreds" of slight attacks. The *slight* attacks which he still had when I first saw him, were so slight that strangers noticed nothing wrong with him; he is never quite unconscious in them; the severest of these slight fits only "bemaze" him for a minute or two; he can go on talking. Here are epileptic attacks with defect ("bemazement"), but not with loss of consciousness. A medical friend who sees much of Quærens observes a little flushing of the patient's face, that he is "as if considering something," but only to his intimate friends is it known that he has any kind of seizure. The only local symptom I heard of is a peculiar feeling in the right hand. In each slight fit he has that variety of the "dreamy state" which I call Reminiscence; this peculiar feeling occasionally occurs in many people who are supposed to be healthy. Quærens quotes Tennyson, Coleridge, and Dickens, about it. I reproduce the quotation from Dickens, and after it the whole of the patient's report of his own case (op. cit.).

> "We have all some experience of a feeling which comes over us occasionally, of what we are saying and doing having been said or done before, in a remote time—of our having been surrounded, dim ages ago, by the same faces, objects, and circumstances—of our knowing perfectly what will be said next, as if we suddenly remembered it."—*David Copperfield.*

"Last year I had the misfortune to become, for the first time in my life, subject to occasional epilepsy. I well remember that the sensation above described, with which I had been familiar from boyhood, had, shortly before my first seizure at a time of over-work, become more intense and more frequent than usual. Since my first attack, I have had only few recurrences of the feeling in question. On two occasions, however, it was followed next day by an epileptic seizure, and I have since treated its occurrence as an indication for immediate rest and treatment.

"There seems to me a twofold therapeutic interest in this experience. First that, whatever pretty suggestions Coleridge and Tennyson may make to account for it, and however universal its occurrence may be regarded by Dickens, it probably ought to be regarded as showing disturbance of brain-function; and that, perhaps, its recognition and removal might sometimes prevent the development of a more important disorder. Secondly, that inquiry in cases of epilepsy may detect a something of this sort, put aside as not being of sufficient consequence to speak of; and yet in truth being a minimised form of *petit mal*, warning to precautions against a larger seizure."

The following is also a striking illustration of slight epileptic seizures with the "dreamy state," before severe fits. A man, H., aged 29, who consulted me, March 1882, began to be ill in 1873 or 1874 (he could not be more precise). He had "curious sensations," "a sort of transplantation to another world, lasting a second or so." He otherwise described them by saying that whatever he was doing at the time he (now I use his words) "imagined I have done this before, imagined I was in exactly the same position years ago." He said too, that it was as if waking from sleep. At first he had these "sensations" at long intervals (he could make statements no more definite), but they became more frequent, two or three a day. He was not quite unconscious in them; he had defect of consciousness only. He thought nothing of them; took no notice of them. Now, suppose he had at this stage consulted a medical man, what would have been said of such seizures? The patient had no crude sensation-warning. I got no more than the facts stated. There might be a natural hesitation to diagnose epilepsy from the "dreamy state" alone, as in this case it was very like, if not quite like, ordinary "reminiscence." I should never, in spite of Quærens' case, diagnose epilepsy from the paroxysmal occurrence of "reminiscence" without other symptoms, although I should suspect epilepsy, if that super-positive mental state began to occur very frequently, and should treat the patient according to these suspicions were I consulted for it. I never have been consulted for "reminiscence" only; there have always been in the cases I have seen, at the time I have seen them, with this and other forms of "dreamy state," ordinary, although often very slight symptoms of epilepsy. Some of the patients who have "reminiscence" with other symptoms in epileptic paroxysms know quite well that its occurrence in healthy people is part of popular knowledge. This case of H. was then, however, most certainly one of epilepsy; the sequel showed it. To go on with the report of his case. One morning (March, 1875) he found his tongue bitten; of anything occurring in the night he knew nothing. He did not consult a medical man until he found his tongue bitten another morning. In February, 1882, he had a severe fit in the day; twice he fell in a fit in public places. His friends told him of other attacks in the day, of which he knew nothing. In them he became unconscious, and after some of them, whilst continuing unconscious, he acted elaborately and strangely.

Another way of showing how slight paroxysms of epilepsy may be, is by the fact that the patient does not mention them when he has severe attacks. This remark applies to slight fits of other kinds. A man (W.) who consulted me for severe fits (and who then had quasi-trifling seizure with the "dreamy state"), came to me a year later, saying that he had had no fits. He had had, however, many slight seizures, but so slight were they that he said no one else knew he had them; he goes on walking in them. His crude sensation is the "epigastric" sensation. I give his own words as to the "dreamy state;" a "double self" and a "thought."—"I get an idea in my head different from what I am thinking of." He might have twelve of these slight fits, for fits they certainly are, a day.

Before leaving this part of my subject I remark, by way of recapitulation, that he who neglects the "dreamy state," because it is indefinite and "merely curious," and such symptoms as chewing, &c., movements, and apparent alteration in the size and distance of external objects, because they seem trifling things, may not even surmise that his patient has the serious disease epilepsy in a rudimentary form, until a severe fit comes to tell him so. Even then it may be said that the slight paroxysms "developed into" epilepsy; but I insist that such slight paroxysms are themselves epileptic. Such slight seizures may be erroneously put down as hysterical, or may be fancifully ascribed to indigestion, malaria, &c.

I have confessed that in former years I have underrated, and even, I find, neglected, the "dreamy state." Both to acknowledge a great clinical fault and for the importance of illustrating elaborate actions *after* epileptic fits with the "dreamy state," I give the following extract. In an article, West Riding Asylum Reports, vol. v., 1875, pp. 116–17 ("On Temporary Mental Disorders after[1] Epileptic Paroxysms"), I mention the case of an epileptic patient who after some of his slight seizures (he had severe ones, too) would act very elaborately. After one he was found "standing by the table mixing cocoa in a dirty gallipot, half filled with bread and milk intended for the cat, and stirring the mixture with a mustard spoon which I must have gone to the cupboard to obtain." But I omitted to state what I find in my notes of this case, that at the onset of his fits the patient had "a sort of dreamy state coming on suddenly." I fear I then thought this symptom too indefinite to be worth enquiring into and recording, or possibly, to adopt Quærens' words, I put it "aside as not being of sufficient consequence to speak of," though I hope the omission was only a blunder. In this patient's case I have no note of any crude sensation-warning.

No better neurological work can be done than the precise investigation of epileptic paroxysms. Whilst epilepti*form* convulsions have been minutely studied, comparatively little attention has been given to the analysis of epilep*tic* fits. Speaking only of epileptic fits and solely of slight seizures of this kind, the endeavour should be not merely to ascertain whether a case is one of "genuine epilepsy" or not, but to describe all that happens in the paroxysm. For although I use the expression "variety of epilepsy" as if there were a clinical entity "Epilepsy," with complications, peculiarities, &c., warranting subdivisions of it, there can be no question that there are at least as many epilep*sies* as there are paroxysms beginning with different "warnings." What we call the warning,[1] this being the first event from, or during, the onset of the local, sudden, rapid and excessive (or briefly the "epileptic") discharge, is the clue to the seat of the "discharging lesion." There are at least as many differently seated "discharging lesions"[2] as there are different warnings of the paroxysms. So that I admit that the grouping together of cases of epilepsy which present, in the paroxysms, the "dreamy state" is an entirely arbitrary proceeding, as much so as taking any other striking symptom to mark a group would be; all the more that not only, as I have said and illustrated, does the "dreamy state" sometimes occur without a crude sensation of smell, &c., but that these crude sensations may occur in slight fits without the "dreamy state." And in the group itself, as arbitrarily indicated, there are at least several different epilepsies; certainly a paroxysm beginning with a crude sensation of smell is a sub-variety, and one beginning with the "epigastric" sensation is another, although in both cases there may be the "dreamy state." The "discharging lesion" must be differently seated in the two cases; most likely the former is a "discharging lesion" in some part of Ferrier's centre for smell. (See also Dr. James Anderson's patient's case, in which there was a "warning" by a taste.) But artificial separations, studies of cases as they approach certain types, are absolutely necessary for clinical purposes. Hence I shall continue to use the expression "variety of epilepsy" for a group of different epilepsies, each of these agreeing in presenting the "dreamy state;" this will not be harmful if we investigate each case on its own merits. But I hope that this empirical method, one much less empirical than the current method, will aid us towards a scientific classification; that we shall ultimately be able not only to speak of certain symptoms as constituting genuine epilepsy or some variety of it, but of these or those particular symptoms as pointing to a "discharging lesion" of this or that particular part of the cortex. This will be trying to do for epilepsy what has been done to a great extent for epileptiform seizures. We may speak of "varieties of epileptiform seizures," but we speak of each case as showing that there is a "discharging lesion" of this or that part of the cortex in the Rolandic region.

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[1] I draw attention to the word "after," as I have been said to adopt the explanation that "epileptic mania" is the outcome of the same degree of discharge as that which produces convulsion. But I hold almost the very contrary doctrine. I have contended strenuously that *elaborate* mental states *never* occur during, and that *elaborate* series of movements *never* occur from (directly from) *such* a discharge. Only such mental symptoms as crude sensations occur during that degree of discharge; such elaborate mental states, as the "dreamy state," in my opinion, *never* do.

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[1] As before said, the "dreamy state" is not a warning in the sense of the word used in the text; crude sensations are warnings in the proper sense.

[2] The expression "discharging lesion" is objected to by some of my medical friends. I have been told that we can understand an ulcer discharging, but not nerve cells. Apart from this kind of criticism, the term may be objected to for better reasons, especially when the word "seated" is used with it. I only mean by "discharging lesion" a vast exaltation of the function (hyper-physiological alteration) of cells of small part of the cortex caused by an abnormal nutritive (pathological) process involving an increased but an inferior kind of nutrition. By "seated" here or there I simply mean that the cells so altered are of this or that part of the cortex. A "discharging lesion," or "physiological fulminate," is an alteration of cells of nervous arrangements of the cortex, representing some impressions or movements, or both, of parts of the body. That the "discharging lesion" (or whatever it is to be called) is often small and local in epilepti*form* seizures (of a few cells of the middle motor centres ("motor region") of one half of the brain) is not, I think, doubtful. I have suggested that the radical cure of fits in such cases is for the surgeon to cut out that "discharging lesion," as well as the tumour, if there be one, producing it. I think too that in epilep*tic* fits the discharging lesion is similarly "doubly local," but of the highest centres. A most excellent name for what I call "discharging lesion" is Horsley's term "epileptogenous focus." My reason for continuing to use the term "discharging lesion" is that it keeps us well in mind that the epileptic process is but an exaggeration, although a vast one, of normal nervous discharges—of a normal *physiological* process.

Before we can make good generalizations we must carefully analyse. To group together as "visual warnings" colour projections, apparent alteration in the distance of external objects and "dreamy states" with definite scenes, is generalising without previous analysis, and is an attempt to organise confusion; they are exceedingly different things. He who is faithfully analysing many different cases of epilepsy is doing far more than studying epilepsy. The highest centres ("organ of mind"), those concerned in such fits, represent all, literally all, parts of the body sensorily and motorily, in most complex ways, in most intricate combinations, &c. A careful study of many varieties of epileptic fits is one way of analysing this kind of representation by the "organ of mind." Again, it is not, I think, an extravagant supposition that there are, after slight epileptic fits of different kinds, many temporary morbid affections resembling those persistent ones produced by destructive lesions of different parts of the cortex. To illustrate for a moment by epileptiform seizures; there is temporary aphasia after some fits beginning in the face or hand (more "elaborate" utterances, I think, when the exact starting-point is in the ulnar fingers); this is the analogue of aphasia from a destructive lesion (softening, &c.) To return to epilepsy. There is, I am convinced, in, or after, certain paroxysms of epilepsy temporary "word-blindness;" certainly in one patient of mine who had a "warning" by noise. I could not make out that this patient was at the same time "word-deaf," but thought his temporary deafness was ordinary deafness. Still there may have been word-deafness. In another patient, who called his attacks "losses of understanding," there was clearly both "word-deafness" and "word-blindness," with retention of ordinary sight and hearing; this patient's attack used to begin with a warning of noise, but he has recently had his "losses of understanding" without that warning.

I have given brief details of some cases of the variety of epilepsy with the "dreamy state" in the preceding introductory remarks. I now narrate other cases at more length.

CASE I.—*Epileptic attacks with crude sensation warnings, by smells in the nose and by the "epigastric" sensation; "Intellectual Aura" or "Dreamy State"; Double Optic Neuritis. Attacks of left-sided tremor—Apoplexy and left hemiplegia. No Necropsy.*

It is well to say at once, that there was no evidence of disease of the digestive, renal, circulatory or respiratory systems. There was no history of syphilis. For most of the notes of the case I am indebted to Mr. Wholey.

A. B., a man 37 years of age was sent from the out-patient room to George Ward, London Hospital, to see me, Nov. 7, 1884. He was subject to attacks of *le petit mal.* The first attack was in 1882; it only lasted about five minutes. He had no more until May, 1884; since which date he had had many, sometimes three or four a week. The attacks began by smells, which he declared to be horrible, but he could give no particular description of them; his wife said that he had likened them to the smell of phosphorus. There was no loss of smell (tested Nov. 17th) as there sometimes is in epileptics who have paroxysms, beginning with such so-called "subjective" smells. (There was no organic disease of the nose.) The patient had another preluding sensation—one seeming to him to start from the epigastric region. No doubt both these crude sensations were concomitant with the onset of the central discharges causing the fits—of the cells of that part of the cortex risen into that high degree of instability which I call a "discharging lesion." His wife said that for a day or two before an attack he felt drowsy and stupid. In the attacks the patient would become "vacant," and would sometimes lose consciousness altogether for a short time. But besides negative affection of consciousness (that is, when consciousness was only defective), there was at the same time the diametrically opposite, the super-positive, state, "increase of consciousness," that is, there was the so-called "intellectual aura," what I call the "dreamy state." Thinking it very likely, because he had a "warning" of smell, that he had this super-positive state, I urged him to tell us all that he felt in his paroxysms, asking no leading questions. He said that he "began to think of things years gone by," "things intermixed [like all the rest on this matter, these were his own words] with what had occurred recently," "things from boyhood's days." Another account is "peculiar sensations passing through his memory and appearing before his eyes." "He thinks of things he has, might, or will do," "he mentally sees people whom he has not seen for some years." He had also in the paroxysms left-sided movements (he was, it is necessary here to say, right-handed). They were described as "trembling." According to the patient's wife, the movements began after the other paroxysmal symptoms; according to him, with them. He said they began either in the leg or in the arm; according to his wife, they always began in the leg. All that is certain is that they were left-sided, but I have little doubt but that they did, most often at least, start in the leg. On January 12, to anticipate, the following note was made:—"Another fit this morning early. Tremor began in the left leg and then 'went up' the side of the body and into the left arm. Trembling was very rapid, and lasted on and off quite an hour. He tried to put bread to his mouth with the left hand, but the trembling prevented him from doing it without using his right hand as well. He tried to walk, and in doing so he says he felt as if he must go to the left, and it was only by dint of a good deal of effort that he could walk at all straight. In some attacks beginning in the left toes there occurred flickering in the left side of the face, and when the arm was gained it was affected after the face." That these attacks should last so long as mentioned in the foregoing note does not invalidate the assertion, that they were owing to central discharges. It is well known that even some severe epileptiform seizures of the common kind last for hours, there being not a mere succession of fits, but one continuous seizure. The "reflexes," superficial and deep, were considered to be normal.

For more than twenty years I have urged the routine examination of the fundus oculi in all cases of nervous disease. But in this case at the first visit I stupidly omitted using the ophthalmoscope. On Nov. 17, Mr. Wholey, my then House Physician, discovered double optic neuritis. On that day I for the first time saw double optic neuritis in a case of this variety of epilepsy. As is exceedingly common in physicians' practice, there was no defect of sight to careful testing. A few days later Mr. Couper examined the patient's fundi, and reported as follows:—

"*Left Eye.*—Considerable capillary redness of disc, with œdematous swelling amounting to 2½ *D.* The choroidal boundary of the disc is concealed from view. The optic nerve fibre bundles are faintly visible above and below. There are radial streaks; they show no blood-staining. The œdema and greyish opacity extend a short way from the disc into the retina. The veins are large and prominent, and slightly varicose. *Right Eye.*—There is more œdema of this disc, the swelling amounting to 3 *D*, also more greyish opacity; the choroidal margin is concealed. The veins are large, very slightly varicose, and this latter change extends far towards the equator throughout several ramifications. There are no visible hæmorrhages or blood-staining of the disc, but there is considerable capillary engorgement, the disc being as red as the adjoining part of the fundus."

Finding double optic neuritis, the conclusion was that all the symptoms were dependent (mostly indirectly) on tumour of the cerebrum, its right half as the left-sided motor symptoms showed. No conclusion was at first come to, as to the nature of the tumour; it was not considered likely to be a syphilitic one.

Under mercurial inunctions the optic neuritis passed off. The fundi were examined again by Mr. Couper, Jan. 5, 1885; the swelling was less. Details of this examination need not be given. On Feb. 19, Mr. Couper examined for the third time, and reported that if he had then examined for the first time he could not have said that there had been neuritis; the discs had become again normal in appearance.

The patient, rid of his optic neuritis, did not seem much better in general. He was slightly bemazed, slightly hesitating, slow rather in speech and suffered from headache. He was, however, up and about, and to an ordinary non-medical observer no decided mental or physical defect would have been at most times observable. But, as often happens in cases of optic neuritis, or, as in this case, in patients who have had it, death occurred rapidly. Mr. Wholey noted, March 25, that the patient had been complaining very much of pain in the head. About 7.30 P.M. the patient went to the lavatory and was no doubt sick there, as some yellowish fluid came from his mouth and nose. He became suddenly pale and fell down unconscious; there was left hemiplegia. He died about six hours after the attack. Necropsy was forbidden.

Commentary on Case 1.—The things to be remarked on are: (1) Double optic neuritis and its existence without defect of sight; (2) Rapid death in cases of double optic neuritis (from the intracranial disease it signifies); (3) Treatment of cases of brain disease with optic neuritis; (4) The left-sided motor paroxysms; (5) Crude sensation-warnings (smell and the "epigastric sensation"); (6) (*a*) Negative affections of consciousness with (*b*) super-positive affection of consciousness. ("dreamy state").

Of 1, 2, 3, and 4, I intend to say little here. I have written on 1, 2, and 3, many times since 1865, and at very great length in the 'Trans. Ophth. Soc.' vol. i. 1881, p. 60 *et seq.* The double optic neuritis is clinically the most important thing in the case, certainly a thing of most importance in prognosis as regards life. It is accepted doctrine now-a-days that it is (*a*) the *best* evidence (which means that it is not decisive evidence) of local gross organic disease (tumour, &c.) within the cranium; that (*b*) it is of no localising value beyond that it points to disease "within the cranium;" that (*c*) it very often exists with good sight;[1] that (*d*) under treatment it may pass off, sight remaining good.

It is certain that double optic neuritis may pass away under treatment when the organic disease within the skull causing it remains, sight continuing good. It is an error to suppose, when a patient is rid of double optic neuritis, of headache, and all other symptoms pointing to intracranial tumour, by anti-syphilitic treatment, that a syphilitic tumour of the brain has been got rid of. No doubt the organic disease in A. B. (Case 1) remained when the optic neuritis had disappeared, and that this disease afterwards caused his death. I always treat optic neuritis in the same way as I should intracranial syphilis; the cause of it is sometimes a syphilitic tumour of the brain; but could I know that the lesion was not syphilitic I should give mercurials and iodides. (Of course, cases in which the neuritis occurs in Bright's disease and cases of swelling of the discs in tubercular meningitis are excepted.) I believe that there would be fewer blind people if the ophthalmoscope were used by routine in cases of severe headache; optic neuritis, discovered in its præ-amaurotic stage, presumably the stage most amenable to treatment, would very often yield to treatment. I do not, however, say that optic neuritis will not pass away without drug treatment. For after removal of tumour from the brain (Horsley), optic neuritis has disappeared when no medicines have been given. Ferrier narrates a case of cerebral abscess ('Lancet,' March 10, 1888) in which optic neuritis passed off after operation by Horsley; no drugs were given. Horsley trephined and evacuated a cerebral abscess, the position of which Ferrier had very accurately diagnosed. The patient is now quite well. Yet I should not dare to omit treatment of the kind mentioned in ordinary cases of optic neuritis. If it does nothing for intracranial tumour, it often, I am convinced, prevents blindness. To return to the case of A. B.

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[1] I very well remember my astonishment on finding for the first time, that a patient with double optic neuritis could see well ("Case of Tumour at the Base of the Brain," 'Medical Times and Gazette,' June 17, 1865; 'Royal London Ophthalmic Hospital Report,' vol. iv. 1865). But now-a-days the young medical men I am acquainted with are astonished that any one doubts that marked optic neuritis often exists with good sight. For all that, although well known, it is not sufficiently known that patients with very striking abnormal changes in the discs may have no visual defect.

It is well known that patients with double optic neuritis often die suddenly or rapidly, as A. B. did. A. B. probably died by hæmorrhage from a vascular tumour of the right cerebral hemisphere (temporo-sphenoidal lobe ?). In some cases, as Hilton Fagge has pointed out, patients who have cerebral tumour die by rapid respiratory failure. This may happen when there is no hæmorrhage from the tumour. It is possible that cerebral tumour, besides producing optic neuritis, sometimes produce similar pathological changes in centres in the medulla, the respiratory among others.[1]

The attacks of one-sided tremor ("diluted convulsion") such as A. B. had, occur, in my opinion, in cases of disease behind the so-called motor regions; if so, there is some little evidence towards showing that the supposed sensory districts are not purely sensory. They were not epileptiform seizures, I mean not like fits I have seen dependent on disease of the so-called motor region. I shall speak of these "diluted convulsions" elsewhere.

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[1] Some time ago ('Trans. Ophth. Soc.' vol. i., 1881, p. 98) Dr. Buzzard said, "Was it possible . . . that the vomiting and slowing of the pulse might represent an affection of the pneumogastric brought about by the same cause as that which produced optic neuritis, and that the sudden or rapid death Dr. Hughlings-Jackson had mentioned as one of the possible contingencies of optic neuritis from intracranial disease, might also be explained by a more severe influence on the same nerve?"

Saying again that the topics 1, 2, 3, and 4 are clinically of vast importance, I shall go on to speak of cases of epilepsy with the "dreamy state" more generally.[1] I have only seen three cases (Dr. James Anderson's patient, A. B., and that of a woman[2]) in which with this variety of epilepsy there were strong symptoms of local gross organic disease within the cranium. Before speaking further of the (5) crude sensations and (6) abnormal affection of consciousness (*a* and *b*), I will narrate other cases of this variety of epilepsy in which there is no reason to suppose that there exists local *gross* organic disease. I say local *gross* organic disease, meaning such as tumours, abscesses, cysts, &c. That there is some local disease in every epilepsy I have no doubt whatever; there is, beyond question, some *pathological* process productive of high instability, which is a functional change (abnormal physiological change) of a few cells of some part of the cortex. I would here refer to remarks I made on the use and misuse of the term functional ('BRAIN,' January, 1888, p. 312).

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[1] The question whether fits of epilepsy are produced by tumour or not is, however, I think important with regard to the epilepsy itself. I imagine that the pathology of most cases of epilepsy proper is that the "discharging lesion" is the consequence of plugging of arterioles (such arterio-cortical pathology is certainly the pathology of some epileptiform seizures). Whether the discharging lesion is produced by an encephalitis about a tumour or by plugging of vessels, the physiological condition (the "discharging lesion") is the same. I am suggesting that there is another sort of difference. Centres for taste and smell lie, accord to Ferrier's localisation, in the region of the posterior cerebral artery, whilst, still according to his localisation, the centres for hearing and part of the centre for sight (angular gyrus) lie in the region of the middle cerebral. Hence, if arterial plugging be the pathology, it may be that we have different varieties of epilepsy proper, according as arterioles are plugged in different vasqular regions. The variety of epilepsy I am remarking on in the text may be owing to morbid changes in the district of the posterior cerebral. But tumours would grow regardless of vascular regions. I suggest that cases of epilepsy with mixed warnings (of smell or taste along with warnings of noise or colour) are more likely to be owing to tumour or other gross organic disease, than to have the minuter pathology I have mentioned—that of arterial plugging and its consequences. However, doctrines as to the sensory localisations are in an unsettled state. Ferrier thinks that another part of the visual centre is the occipital lobe, and this region is supplied by the posterior cerebral artery. Schäfer (BRAIN, April 1888) says that the visual area "comprises the whole of the occipital lobe," and that it "perhaps includes a part or the whole of the angular gyrus."

[2] See footnote 1, p. 179.

The case next to be related is unusual, in that the severe fits preceded, for some years, the slight attacks. It is, I think, exceptional in other ways.

CASE II.—*Severe epileptic fits without "warning" for twenty-five years. —Slight seizures for the last eighteen months (true vertigo; smells; "dreamy state.")*

William B., aged 47, was an out-patient under the care of Dr. Beevor, National Hospital for the Epileptic and Paralysed.

My colleague, knowing my interest in this variety of epilepsy, told me the particulars of the patient's fits and permits me to report the case. It may be well to give first what the patient, at my request, wrote down of his own case, exactly in his own words. "W. B——, who for twenty-five years suffered from occasional attacks of epilepsy, has now for the last eighteen months (on and off) been subject to [attacks of] violent giddiness and headache, accompanied with strange smells and tastes, the more prominent being that of chloride of lime; he also has at the same time, the sensation of walking or moving in space and being brought into close contact with prominent buildings, such as churches, railway stations, &c., although a long distance from them, and not having seen them for a length of time."

I now give a fuller account, which I got by enquiry, although it involves some recapitulation. For twenty-five years the patient had been subject to fits, in which he became unconscious and was convulsed; his tongue was not bitten. Of these attacks he had no immediate warning, but was drowsy six or eight hours before their onset. He would not go to town on business when he felt drowsy, taking this as evidence that a fit was coming. He had at that time no slight fits. For eighteen months he has had slight fits only, and it is to the peculiarities of these that I wish to draw attention.

The first thing is a giddy feeling; it is true vertigo. This case is the only one investigated by myself in which I have been certain of the occurrence of this variety of vertigo—apparent movements of external objects—in epileptic fits with the dreamy state (p. 108). He said "things were all of a move," and was quite sure that they moved to one side; he was not absolutely certain to which side they moved, but was almost certain that they passed to the left (apparent movement of real external objects is meant; not of ideal objects, *vide infra*). With or just after this came "strange smells and tastes," the "most prominent" being like chloride of lime; he said it was odd to speak of the taste of chloride of lime as he had never tasted that substance. He knew nothing of chewing, &c., movements, and had not been told of them. The next thing was his "dreamy state." He seemed to actually see large buildings which he had once seen; it might be that he seemed near a church, "close to its wall." In the last attack he "saw" certain almshouses, "all in a moment saw that building and could actually see the clock." The things he "saw" seemed of a natural colour.

He did not always lose consciousness; when he did it was just after "seeing" the buildings. Indeed in one, since the above was written, he had "had the smell," but no movements of external objects and no visions. The general feeling in the fit was disagreeable; he said there was no fear. There was no epigastric sensation. He did not pass urine in the attacks. There was no tendency to evacuation of the bowels after them. He had no one-sided symptoms, beyond the feeling of displacement of external objects to one side. He was not left-handed.

His smell, each nostril, was tested by Dr. Beevor and was found present. His taste was not defective so far as he knew. He was, he said, always sniffing; I observed that he was always doing it when with me; it was like some one strongly inhaling a scent. He had a sensation of trickling in the left nostril, but there was no unusual secretion from the nose. His optic discs were normal. He could not hear quite so well with the left ear as with the right, but the defect was trifling.[1]

Dr. Ferrier has given me the notes of a case of epilepsy, in which there was a crude sensation of smell with the "dreamy state." In the letter accompanying his report, he writes, "I send you the notes of the case of 'reminiscence' which I promised you. I think you will find it interesting in reference to your views of 'dreamy states.' The patient described the stages of her condition with great lucidity, and without any suggestions from me." The following is the account which Dr. Ferrier has sent to me. The case is, I think, unusual in that the smell comes last.

CASE III.—*Attacks of le petit mal, &c.; Crude sensations of smell and the "dreamy state;" Reminiscence.*

X., married, the mother of three children, had a convulsive attack (said to have been uræmic eclampsia) about a year ago, which came on while nursing her husband in an attack of scarlet fever. Since then she has been subject to attacks of *le petit mal*, without convulsions. These attacks coincide with her monthly periods, and always begin in the night and continue for a day or two. They vary in frequency; occasionally as many as seventeen occur in one day. They are always of the same character, and the patient describes them as going through three distinct stages. The first stage is a dreamy state or reminiscence, in which everything around her seems familiar or to have happened before. The second stage is a pain in the stomach; and the third is a "terrible" smell in the nostrils, associated with a similar taste and occasionally nausea, but without vomiting. Then she feels tired and sleepy. She never falls during the attacks, but says her husband knows when she has an attack by a kind of imploring look on her face, as if she were asking for help.

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[1] As said, warnings by crude sensations of smell occur in epileptics who have no "dreamy state." Recently Dr. Beevor has drawn my attention to the case of a woman (L. E.) who has that warning and no "dreamy state;" she has another crude sensation, a peculiar feeling in the two ulnar fingers of the left hand.

When the attacks occur in the night she knows of their occurrence and their number by being awakened each time by the terribly offensive smell.

The patient is spare (formerly robust) and rather anæmic. There are no indications of organic disease anywhere. The urine is free from albumen; there are no indications of local affections of the nostrils.

CASE IV.—*Slight epileptic attacks for many years ("Dreamy state," &c.), before severe fits; onset of severe attacks by feelings like those of the slight fits—Certain movements of lips.*

A woman, M. W., 42, consulted me, 1881. Since the age of 13 or 14 she had been subject to the frequent occurrence of slight "nervous attacks"; with each of them was "reminiscence." She also called them "flashes of unconsciousness;" the two descriptions showing that the mental state was duplex, of two opposite elements. For many years she took no notice of them, never mentioned them, nor did any other person observe anything wrong. At length she consulted a medical man, who said that her case was one of hysteria. As the sequel showed, this was not so. Two years before I saw her, when her age would be about 40, she had two severe epileptic attacks with tongue biting, and a third later on. Of course it may be said that she may have had the two severe epileptic attacks quite independently of the "nervous attacks," and that the two things were quite different. But she continued to be subject to the "nervous attacks," and that they were slight fits of epilepsy, or, if any one likes, abortive fits, is shown by the fact, that each of the severe attacks began by her having in a slight degree the feelings she had in the slight attacks.

The slight attacks might occur two or three times a day, or she might miss a month. They are very slight. As said, no one, early in the case, noticed anything of them. Of recent years, only once has any one, other than her intimate friends, noticed anything wrong, nor do they always. If walking, she goes on walking; only once or twice have they been so bad that she has had to stop. If at tea, she would go on pouring out, but would pour out wrongly. She may go on with her sewing, and may thread a needle. These things show that her attacks are very slight. (*Vide* p. 183, on negative affection of consciousness.) The feeling she describes in the slight attacks used to be pleasant, but is not so now: there is, however, she says, no fear with it. The attack is "over in a moment." Her "dreamy state" is variously described as "like some scene or dream," "as if you remember that again." The feeling is vivid, and yet she cannot say what the particulars of the "dream" or "scene" are. It is not of anything which has happened in real life, but is like what has happened (I suppose she meant that the "dreamy state" was as vivid as if it were a memory of something which had actually happened). So much for the positive, or rather super-positive, element of her mental condition in the seizures. She speaks of a negative element, "a withdrawal from the present; she hears people talking, but does not know what they say."[1] She may, she is told, reply, but gives random answers. There is clear evidence of defect of consciousness; her expression "withdrawal from the present" is a popular expression, meaning the same thing as "defect of (object) consciousness"; I say *defect*, because I take it that the withdrawal was, considering the facts stated when dealing with the slightness of her attacks, partial only. As a rule she does not lose consciousness altogether.

She does not pass urine in the slight attacks; there is no tendency for her bowels to be moved after them.

As to crude sensations. Sometimes when the attacks "are about" (occurring frequently) she has a sensation either of smell or of taste at the back of the throat, *but not in connection with the seizures.* A friend who came with her said that M. W. would make, in the seizures, a peculiar movement of her lips, slightly like a tasting movement. There was no evidence of one-sided symptoms, that is, no account of spasm, numbness &c., on one side of the body.

I ask a similar question to that I asked when giving some details of the case of H. (in the introductory remarks, p. 186), what would have been thought of this patient's "nervous attacks" if she had consulted one for them before she had the severe fits? If they were like those she has had since the severe ones, one could have concluded that they were epileptic. But such a diagnosis, say it had been made when she was 14, would have been accepted by few medical men, and by some the non-occurrence of ordinary epileptic attacks for years would have been taken as proof that the "nervous attacks" were not epileptic. That she was epileptic from the age of 13 or 14 I have no doubt whatever.

CASE V.—*Slight attacks of Epilepsy with the "dreamy state" for some years before severe attacks—Mouth movements—Automatic actions during unconsciousness (which continued after the slight fits).*

The following is a very important case. It is that of a highly educated medical man, who reports it himself. Names of places are omitted or altered from his original report, the alterations being endorsed by the patient; the alterations make no difference in the medical import of the case. He had first very slight attacks, then severe attacks at long intervals also. I shall comment on the slight attacks only.

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[1] It is easy to say of this one part of the condition (common in slight attacks of epilepsy) that it is "word-deafness." If it be, it is, or, rather, is part of, a negative affection of consciousness.

What he calls "recollection" is what I have called "reminiscence." I retain his term "aura," putting it between commas, although I do not use it myself for any form of the "dreamy state." The report shows clearly that he has some attacks without *loss* of consciousness (see his remarks on reading poetry and on his glacier expedition). In other attacks he had loss of consciousness, and during unconsciousness continuing after them he acted automatically. The actions related in the closing paragraphs show very complex, special, &c., actions after a fit which was presumably slight. I may refer to remarks on this matter in my part of the discussion on Dr. Mercier's paper on "Inhibition," which will appear in some future number of this Journal.

He had no crude sensation, but the words I have italicised in his account of his physical state, p. 204, during the slight paroxysms imply, I consider, discharge of cortical elements, serving during taste. (The report was finally sent in July 1888.)

"I first noticed symptoms which I subsequently learnt to describe as *petit mal* when living at one of our Universities, 1871. I was in very good general health, and know of no temporary disturbing causes. I was waiting at the foot of a College staircase, in the open air, for a friend who was coming down to join me. I was carelessly looking round me, watching people passing, &c., when my attention was suddenly absorbed in my own mental state, of which I know no more than that it seemed to me to be a vivid and unexpected 'recollection';—of what, I do not know. My friend found me a minute or two later, leaning my back against the wall, looking rather pale, and feeling puzzled and stupid for the moment. In another minute or two I felt quite normal again, and was as much amused as my friend at finding that I could give no distinct account of what had happened, or what I had 'recollected.'

"During the next two years a few similar but slighter attacks occurred, involving mental states which struck me as like to the first and to each other, but of which I can now recollect no details. I asked medical advice, but gathered no explanation, received no treatment, and regarded the matter playfully as of no practical importance. I have been in the habit of dreaming very little all my life, but during these years noticed a few occasions when I woke in the night with an impression that I had succeeded in recollecting something that I wanted to recollect, but was too sleepy to give any attention to it, and had no definite idea of it in the morning. These feelings were slightly uncomfortable, and usually, I think, accompanied by a slight involuntary escape of saliva found on the pillow in the morning, and once or twice by a soreness of the edge of the tongue, due, I should presume, to its having been slightly bitten. They did not recur after about 1875.

"In 1874 I first had a *haut mal*, preceded by the mental condition I had felt in *petits maux*, and after medical advice from a physician in London learnt the nature of the disease, and began to attend a little more carefully to the symptoms, which interested me more, as I had then begun to turn my attention to medicine.

"I had a severe attack of pneumonia with pleurisy, and perhaps empyema, beginning in October, 1875, and during slow convalescence (Dec. 1875—March 1876) was more frequently affected. The character of the *petits maux* gradually became more stereotyped, and during the period 1876–1886 varied only within comparatively narrow limits. I will attempt to describe the features which I think were common to all, or nearly all.

"*Mental Condition.*—In a large majority of cases the central feature has been mental, and has been a feeling of Recollection, *i.e.* of realising that what is occupying the attention is what has occupied it before, and indeed has been familiar, but has been for a time forgotten, and now is recovered with a slight sense of satisfaction as if it had been sought for. My normal memory is bad, and a similar but much fainter feeling of sudden recollection of a forgotten fact is familiar. But in the abnormal states the recollection is much more instantaneous, much more absorbing, more vivid, and for the moment more satisfactory, as filling up a void which I imagine at the time I had previously in vain sought to fill. At the same time, or perhaps I should say more accurately in immediate sequence, I am dimly aware that the recollection is fictitious and my state abnormal. The recollection is always started by another person's voice, or by my own verbalised thought, or by what I am reading and mentally verbalise; and I think that during the abnormal state I generally verbalise some such phrase of simple recognition as, 'Oh yes—I see,' 'Of course—I remember,' &c., but a minute or two later I can recollect neither the words nor the verbalised thought which gave rise to the recognition. I only feel strongly that they resemble what I have felt before under similar abnormal conditions. I re-enter the current of normal life, as a rule, quickly—sometimes, as far as I can judge from my own movements or other people's evidence, within ten or fifteen seconds; there is never, however, as sudden a rush of returning normal consciousness as there has been of incipient abnormal consciousness; it is more gradual, and it is hard to say when it is complete, as it almost always leads up to a passive and non-critical mental attitude, in which I feel no originative mental impulse. One point which I almost always feel a tendency to avoid, though I am generally dimly aware of a previous wish to attempt it, is to go over my previous abnormal mental state critically and to give my attention to all its details. But attention seems not to be completely under my control; I sometimes put it off, and delude myself with the impression that remembrance will be just as complete after another five minutes, sometimes let it slip with a feeling of indifference, and sometimes, if I am in company or in any active employment, I have no distinct recollection of any desire for self-criticism or analysis. Accompanying this want of control over reflection I often notice a temporary loss of memory for habitually familiar names or facts, which lasts a minute or two, or sometimes more, after my consciousness seems otherwise normal. This may co-exist, indeed, with so normal a state of consciousness, that I can hardly believe I shall find any difficulty in saying what I want to say, and so I fall now and then into the mistake of beginning without hesitation a sentence which I cannot finish. I have found myself just after a *petit mal* at a London Railway Booking Office, meaning to go to K——, and asking without hesitation for 'Second return to—to—that school, don't you know—' (or some such words) and being a good deal startled at my forgetfulness.

"A *petit mal* has two or three times come on when I have been reading poetry aloud—the line I am reading or just going to read seems somehow familiar, or just what I was trying to recollect, though I may never have seen or heard it before. I recognise my morbid condition and stop, though I have generally sense enough to finish the line or even sentence, and remain silent for a minute or so; then go on again where I left off, recovering my sense of rhythm and metre sooner than my capacity of giving attention to or understanding the words. I do not remember to have made any deliberate effort to go on reading aloud, *coûte que coûte*, throughout a *petit mal*. I have made several rude attempts to go on writing, and have kept four or five specimens of what I have written. They were made in very slight *petits maux*. The writing was done slowly and in a fairly normal hand. I was in the main occupied with the usual impression of recollection, but was dimly aware that I was morbid, and attempted to criticise what I was writing. My impression at the time that I was writing was that the words and sense were quite reasonable, and that I had kept within very familiar and prudent limits of expression. I had found, I thought, just the words I was seeking for. A minute or two later I could see that some of the words were grotesquely *mal à propos*, though I think the grammatical forms of sentence were always preserved. I could not trace any undercurrent of thought or recollection from which the irrelevant words had come.

"*Physical Conditions.*—As to the physical conditions accompanying these mental states I can gather a little from my own consciousness, and have learnt a little more from friendly observers. At the onset I can rarely notice any physical change in myself, my attention being chiefly occupied with my mental condition; but once or twice when I have been standing near a mirror I have noticed pallor of the face, and I have learnt from others that this is common, and that my eyes have a somewhat staring vacant look as if they were not directed to anything near me, or indeed taking notice of anything particular. In this condition I am told, and in fact occasionally remember, that I often say 'yes,' with an air of complete assent to any remark made to me, whether it is a pertinent answer or not; and further, that I occasionally make a slight half-vocalised sound, whether addressed or not. This latter, I have been told, *is somewhat like a modified and indistinct smacking of the tongue like a tasting movement, and is generally accompanied by a motion of the lower jaw*, and sometimes by some twitching of the muscles round one or both corners of the mouth or of the cheeks, but by no sense of taste in my recollection. I have no clear evidence that one side of the face is affected more than the other, and no clear evidence against it; from what little I can learn, if it is at all unilateral it is rather more on the right side than the left; but the evidence is very scanty. I never notice it myself. I also never notice myself, but learn from others, that sometimes, especially if sitting, I give one or two light stamps on the floor with one foot; and in the only cases where this has been accurately observed it has been with the right foot.

"With the returning normal consciousness I generally feel some superficial flush over the skin, especially over the face, and a slightly quickened and more thumping heart-beat which does not go beyond causing me very slight *malaise*. A very constant symptom is increased urinary secretion, which sometimes makes itself felt in as short a time as five or ten minutes, but usually after a longer interval. The water, if soon passed, is very light in colour, of low specific gravity, once or twice as low as 1005, and contains no albumen.

"The *petits maux* have not been accompanied or followed by hallucinatory sensations of sight, sound, taste, smell or feeling. There has been, I think, no loss of balance. I well recollect in 1878 running across a Swiss glacier, and jumping across many small crevasses when the initial stage of 'aura' came on, and a reflection shot through my mind, that if ever I was likely to pay dearly for the imprudence of going on, it would be then. But I had insufficient control to stop myself and felt no fear, but only a slight interest in what would happen. I went through the familiar sensations of *petit mal* with such attention as I had to give concentrated on them, and not on the ice, and after a few minutes regained my normal condition without any injury. I looked back with surprise at the long slope of broken ice I had run over unhurt, picking my way, I know not how, over ground that would normally have been difficult to me. In the same way a *petit mal* when I was playing lawn tennis did not in the opinion of my adversary make my strokes or judgment of pace and position of balls to be struck any worse than normal. I had no recollection of the strokes during a minute or two.

"I had no *haut mal* before 1874, and since then such attacks have recurred mostly at long intervals, sometimes of as much as eighteen months; during slow convalescence from pneumonia, however; in 1875–6 I had as many as seven or eight in two months. The 'aura' of recollection has preceded all of them, more or less, but is less vivid in my subsequent memory than after a *petit mal.* My evidence as to the subsequent phenomena of the *haut mal* is very incomplete. My loss of consciousness has not seemed longer to those who watched me more than five or ten minutes as a rule, but my loss of memory has been longer and my return to consciousness more gradual. I have not heard that there has been any epileptic cry; the muscular spasms have been variable but generally slight, and not specially localised (except that once I was told of a constant grasping motion of my right arm and hand). In one or two cases the spasms have not been noticed, and the state has been at first supposed to be one of syncope; but some snoring has almost always been noticed before recovery. My subsequent mental condition has been one of indifference and a sense of fatigue; my bodily sensation is, as a rule, of having been lightly bruised all over.

"During the past year (1887), and more especially during the last four months, there has been some change in the symptoms of the *petits maux*, which may be shortly summed up by saying, that there has been less vivid sense of recollection and there have been longer periods of automatism without memory. I think I had best attempt to explain what I mean by two or three instances.

"(1.) In October 1887 I was travelling along the Metropolitan Railway, meaning to get out at the fourth station and walk to a house half a mile off. I remember reaching the second station, and I then recollect indistinctly the onset of an 'aura,' in which the conversation of two strangers in the same carriage seemed to be the repetition of something I had previously known—a recollection, in fact. The next thing of which I have any memory was that I was walking up the steps of the house (about half a mile from the fourth station), feeling in my pocket for a latch-key. I remembered almost at once that I had had a *petit mal* coming on at the second station, and was surprised to find myself where I was. I recollected that I had meant to reach the house not later than 12.45, and had been rather doubtful in the train whether I should be in time. I looked at my watch and found it within a minute or two of 12.45. I searched my pockets for the ticket, which was to the fourth station, found it gone, and concluded that I must have passed the third station, got out at the fourth, given up my ticket and walked on as I had previously intended, though I had no memory of anything since the second station some ten or twelve minutes previously. I imagine that I had carried out my intention automatically and without memory.

"(2.) Again, in November 1887, after dark—about 6 P.M.—I was walking westwards in a London street, when I felt a *petit mal* coming on of which I can remember no particulars. My intention was to walk westwards for about half a mile; my thoughts were occupied with some books I had been reading in a house which I had just left. With my return of memory (which was incomplete and indistinct) I found myself in a street I did not at first recognise. I was somewhat puzzled, and looked up at the street corners for information as to the name of the street. I read the name "P—— St." which crossed my path at right angles, and with some difficulty realised that I was walking not westwards, as I had been intending, but eastwards, along the street by which I had come, and had, in fact, retraced my steps some three hundred or four hundred yards. I felt no purpose in doing this, no aim at going anywhere in particular, and to save further difficulty, and because I was puzzled, I got into a hansom which was standing still close by me. I have no recollection of giving the driver any orders, and was in a very unreflective state. My impression is that the cab-driver drove quickly to the right house, and I distinctly remember some slight surprise I felt at his knowing the house, and at finding myself giving him a shilling, when I doubt if I could have explained where he came from. Immediately after entering the house I realised tolerably distinctly what had probably happened, and looking at my watch, I calculated that I had not lost more than five minutes by this, if so much.

"(3.) About a fortnight later I was walking by the same route about 10.30 P.M., and again felt a *petit mal* at a point within a hundred yards or so of the one described above. I cannot be certain that a memory of the previous attack recurred to me, but I think it is very probable. My memory again was a blank until I found myself facing eastwards and looking up at the name "P—— St." Then the memory of the previous retracing of my steps recurred to me at once. I more quickly than before gathered together full consciousness, felt a cab unnecessary, walked home, and had no difficulty in writing steadily for about three hours without fatigue.

"In the earlier of this pair of cases (2 and 3) I had no thought whatever of going back to the house where I had been reading, or to any point in that direction; but I believe I am correct in saying that I was thinking of what I had just been reading there. As far as I know, this is the first instance of my changing my intended action *ex proprio motu* in a mental state of which I have no memory. In the companion case (3) I cannot feel sure how much I was influenced by recollection in the earliest stages of the *petit mal*.

"(4.) A fourth occasion is perhaps worth record. I was attending a young patient whom his mother had brought me with some history of lung symptoms. I wished to examine the chest, and asked him to undress on a couch. I thought he looked ill, but have no recollection of any intention to recommend him to take to his bed at once, or of any diagnosis. Whilst he was undressing I felt the onset of a *petit mal*. I remember taking out my stethoscope and turning away a little to avoid conversation. The next thing I recollect is that I was sitting at a writing-table in the same room, speaking to another person, and as my consciousness became more complete, recollected my patient, but saw he was not in the room. I was interested to ascertain what had happened, and had an opportunity an hour later of seeing him in bed, with the note of a diagnosis I had made of 'pneumonia of the left base.' I gathered indirectly from conversation that I had made a physical examination, written these words, and advised him to take to bed at once. I re-examined him with some curiosity, and found that my conscious diagnosis was the same as my unconscious,—or perhaps I should say, unremembered diagnosis had been. I was a good deal surprised, but not so unpleasantly as I should have thought probable."

(To be continued.)

ON PARALYSIS BY EXHAUSTION.

BY CH. FÉRÉ, M.D. (PARIS),

Physician to the Bicêtre Infirmary.

PARALYSES by exhaustion, that is to say, due to an excessive nervous discharge, local or general, may arise under various conditions. Those observed after epileptic seizures have received a considerable share of attention, and vary from a simple localised weakness to a complete and diffused paralysis.[1]

Briquet[2] has noted that hysterical convulsive attacks may give rise to loss of muscular power, and that the paroxysms often increase pre-existing palsies. Among other causes of hysterical paralysis he mentions strong emotions, excessive fatigue, forced marches, prolonged work, among washerwomen and harvest girls for instance, exhausting evacuations. "I have seen," he says, "paraplegia follow diarrhœa of six months' duration; and paraplegics suffer, after menstruation, from regular increase of the symptom lasting seven or eight days." These paralyses, due to malnutrition of the nerve elements, have been observed by several other authors, especially by Abeille[3] and Landry,[4] and may be mentioned here, inasmuch as they occasionally originate in a mental shock. But it is not these which are occupying us just now.

The paralyses I wish to discuss are due to voluntary movements unduly prolonged, involving a local exhaustion, the manifestations of which are more or less diffuse.

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[1] Ch. Féré, "Note sur l'état des forces chez les épileptiques" ('Bull. Soc Biologie,' 1888, p. 24).

[2] 'Traité de l'Hystérie,' 1859, pp. 443, 444.

[3] 'Étude clinique sur la Paralysie indépendante de la Myélite,' 1854, p. 100.

[4] 'Recherches sur les Causes et les Indications curatives des Maladies nerveuses,' 1855, p. 41 ff.

CASE I.—P., aged 32, ironsmith, attended the Salpêtrière consultation on April 20, 1885, being affected since the day previous with right hemiplegia, chiefly in the arm. No known neuropathic family antecedents. Nocturnal terrors when young; chorea at about seven, lasting six weeks, after being frightened by a bull. No rheumatism nor other illness. He served in the army for five years, and always enjoyed good health. Afterwards worked regularly as smith. Unmarried; no excesses of any kind; takes fairly good food.

Two days ago he had two hours extra to finish some work. About one hour before leaving off he noticed that the hammer felt unusually heavy, and on going home his hand felt as if numb. Feeling very tired, he went to bed without eating, and slept calmly and soundly. On waking up yesterday morning his right arm was hanging powerless and flabby; and on trying to get up his right leg gave way under him. He called through the partition a friend in the next room, who rubbed him energetically with *eau sédative*, and so restored some power at least in the leg. He was able to stand, and even to go down-stairs, though with dragging of the foot; the arm remained powerless. Patient had no pain, no fever, no gastric trouble. His state was unchanged till the Monday morning, when he went to the consultation.

On examination, he was found to be a tall, fair man with good muscles and white skin. Very few hairs on the face; mucous membranes pale, sclerotics bluish; anæmic sound at the base. No other abnormal sign in the chest, no defect of conformation.

On the left side the labial commissure is somewhat drawn upwards and sidewards, the palpebral aperture slightly narrower, and the nasolabial furrow deeper. No deviation of tongue.

The right arm is completely flaccid and hanging helplessly, incapable of any movement from the shoulder downwards, forwards, or laterally; the forearm is also incapable of any movement. Slight extension and flexion of the four fingers together are the only movements in the hand; the thumb quite immovable.

The patient has been able to walk two kilometres (nearly one and a half mile), to come from his lodging to the hospital, as the functions of the leg are far better preserved than those of the arm. The movements of the lower extremity are all present, though weaker. Flexion of the thigh, leg, and foot is incomplete on walking; there is dragging of the external border of the foot. Patient experiences unusual fatigue.

General sensibility is abnormal on the right. On comparison of the reactions of symmetrical points to contact, temperature, pinching, pricking, right hemianæsthesia becomes apparent, though not uniformly spread over the whole of that side. The face reacts nearly normally to contact, the trunk and leg much less, the arm and shoulder hardly at all, and do not bleed to deep puncture. Patient can move his right leg in the dark, and perceives changes of position given to it; he is not aware of the position of his right arm unless he sees it. Special senses apparently intact; some peripheral diminution of visual field, and amblyopia, though no achromatopsia, of right eye. Right knee jerk slightly augmented. Some tenderness to pressure in left testicle; and to tickling and pressure over spines of last three dorsal vertebræ.

The characters of this paralysis, involving both feeling and motion, coming on gradually without injury, and the presence of some hysterical signs with anæmia, made one diagnose a functional hemiplegia. I advised the patient to carry his arm in a sling, and live in the open air, without fatiguing his weak right leg, and prescribed cold douches, with iron and strychnia before the meals.

On the 27th of April the only sign left in the leg was a slight exaggeration of the knee-jerk. Face asymmetrical; visual field normal. Sensibility equal on both sides in face, leg, and trunk. Right arm much as before. The anæsthesia occupies the whole region of the shoulder, scapula, and anterior half of the clavicle. On the left side the dynamometrical pressure is 52. Treatment continued, with massage of all teguments and passive movements of arm for five minutes evening and morning.

May 4.—Arm much improved; can be raised to 30° with body; elbow can be moved forwards and backwards to level of thoracic plane, or a little beyond. Flexion and extension of fore-arm possible where unresisted; very little pronation and supination. Thumb movable in every direction. Fingers move awhile also; but closure of fist still incomplete and weak. Dynamometer: left 50 kilos, right 12. Anæsthesia occupies the same cutaneous area, but is less intense; patient recognised the general direction of the limb, though unable to define its exact position, and its exact removal from the body.

May 11.—Only slight difficulty in raising arm above horizontal line. Movements of hand and fore-arm normal. Dynamometer: R. 56, L. 48. Cutaneous and deep sensations have returned. Testicular and dorsal tenderness still present. Patient intends to resume work to-morrow. Advised to continue treatment; but has not attended hospital since.

The evolution and determination of this paralysis confirm the diagnosis of a dynamic disturbance, whilst the distribution and character of the anæsthesia bring it into relationship with the hysterical monoplegias lately described by Prof. Charcot. In the following case the hysterical element is still more evident.

CASE II.—Mdlle. D. has a neurotic family history. Paternal uncle insane; mother suffers from violent migraines and various hysteroid symptoms; two younger brothers said to have died of meningitis when infants.

She has had early convulsions, and from 11 years of age hysterical accidents, when I treated her for rhythmical chorea of three months' duration, following mental shock. I then found ovaria, with slight anæsthesia on the left, with painful spots along spine and on vertex.

She is good-looking, and her expression and physical development are in advance of her actual age (14 years). She has fair hair, white skin, and is well nourished. Menstruation regular and painless since eighteen months ago. Some anæmia and palpitations; otherwise healthy. No taste for any study except that of piano playing, which she carries on to excess.

Last July 19th, the day before her grandfather's birthday, which she was to celebrate by a performance, she practised for nine hours, till late at night, and even then she went to bed only because her left hand felt heavy and clumsy. Whilst undressing she found that she was obliged to look at the hooks and buttons which her fingers no longer felt. (She had already, under similar conditions, noticed such phenomena, though the next morning she used not to feel anything of it.) On being awakened as usual at 8 A.M., she was surprised to find her left arm anæsthetic and completely paralysed. The left leg could be moved, but was weak and heavy.

July 20.—At 11 A.M. I found the patient perfectly cool and collected, to the great astonishment of her friends, notwithstanding the trying circumstances under which she found herself. No facial deviation; left arm motionless and relaxed; slight movements possible of the upper arm from the shoulder, but hardly any in fore-arm and hand.

When patient is lying down, the foot is rotated outwards; when standing she leans on the right leg, and when walking drags the outside border of the left foot; the weak leg partly giving way under her, and standing on the latter is difficult, and becomes impossible when the eyes are closed. The gluteal furrow is somewhat effaced; but the knee-jerk remains normal.

No recent diminution of sensibility, general or special, on the left side of the face. Some anæsthesia of the leg, in which she preserves however the sense of position, though unable to stand on it with the eyes closed. The arm is in its lower third entirely anæsthetic to contact, pinching, and temperature. This condition gradually shades off into the permanent deficiency of sensibility that still obtains about the shoulder. The patient feels when her arm is moved forwards, or backwards, or away from the trunk; but is unconscious of movements communicated to the fore-arm, wrist, and fingers. She recognises a change of position in the limb only by movements of the shoulder. No cutaneous discoloration, nor change of temperature in the left hand.

With the eyes closed the patient cannot form any idea of her left hand whilst the fingers are being flexed and extended. She can represent to herself the most complicated movement of the right hand; but on the left side the limb seems as if lost in a fog, and its form cannot be pictured in her mind.

The patient was kept in bed, cold affusions being applied night and morning, and strychnia and iron given with each meal, with 30 grains of bromide at night.

On July 23rd, four days after the seizure, no change was noticeable. Passive movements of all the joints of both arms alternatively were practised night and morning for ten minutes. On the 26th only some signs of flexion of the left thumb were noticed: these movements were much more apparent when accompanied with similar active or passive movements of the right thumb, or whilst the patient saw them practised by another person. The other fingers followed suit.

Dynamometric pressures:

		Right.		Left.
July 28	...	26	...	10
„ 29	...	25	...	10
„ 30	...	25	...	12
„ 31	...	23	...	14

After repeated flexions of the right hand these last two numbers reached 24 and 16 respectively, and on August 2nd they were 21 and 17; all the movements of the hand and arm had returned, first in the lower, then in the upper part of the arm. Since then no increase was observed in the dynamometric pressures, which seem to have reached their normal level. Miss D. resumed her musical studies two weeks after her restoration to health, and has not felt any abnormal symptoms. The mental representation of movements is the same for both hands.

It is not necessary to insist upon the numerous analogies between these two cases. In both we find the same cause, viz. exaggerated exercise. The local distribution of the symptoms, paralytic and anæsthetic, is the same; they run a similar course towards a similar end. A point that has not yet been sufficiently studied is that, as dynamometric explorations show, the muscular power is greater on the healthy side during the paralytic stage than after recovery. Though the difference is not considerable, it deserves consideration, inasmuch as I have noticed the same phenomenon in paralyses by hypnotic suggestion.[1] In organic hemiplegia, on the contrary, there is generally a diminution of muscular power on the healthy side (Pitres, Dignat, Friedlaender). It will be interesting to find out whether this augmentation is usual in functional paralysis. The general increase of muscular tonicity on the opposite side of the body might perhaps explain the extreme facial deviation shown by Prof. Charcot[2] to occur in some cases of hysterical hemiplegia, and giving the appearance of a contracture on the side opposite to the paralysis of the limbs.

It has been observed[3] that when the so-called neuromuscular hyperexcitability is brought into play upon the flexors of the fingers, whilst the certain hysterical subjects have a dynamometer in the hand, an increase of power is manifested (10 to 20 kilos) on that side, whilst a diminution takes place on the other (17 instead of 21 kilos). This phenomenon of compensation appears to be of the same nature as that which I have just been discussing.

But the most interesting peculiarity of our two cases is without doubt the etiological factors. Since Prof. Charcot has experimentally demonstrated the important part played by suggestion, or idea, in hysterical paralyses, and particularly those of traumatic origin, there is a tendency to explain on the same principle all functional paralyses consequent upon shock. Though in our cases fatigue or exhaustion, not shock or suggestion, appear to be the cause, still they seem to me to throw light upon the problem.

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[1] 'Sensation et Mouvement,' p. 28.

[2] Bristaud et Marie, "De la déviation faciale dans l'hémiplégie hystérique," ('Progrès Médical,' 1887, vol. i., pp. 84, 128).

[3] Descubes, "Étude sur les Contractures provoquées chez les Hystériques à l'état de veille:" 'Thèse de Bordeaux,' 1885.

We have seen that Briquet and other authors attribute some forms of hysterical paralysis to exhaustion; others have also mentioned fatigue with reference to the so-called paralyses through idea: thus in the remarkable case of Russell Reynolds[1] this factor precedes the evolution of the psychical process.

Excessive exercise of one arm appears to give rise to hemiplegic symptoms. Thus Frank Smith,[2] of Sheffield, describes as "hephæstic hemiplegia, or hammer palsy," symptoms observed chiefly among men who have to wield rapidly a small hammer. The disease affects chiefly anæmic individuals, and is most marked in the arm; the face and leg are also involved, and sometimes there occur also aphasia, ptosis, general anæsthesia, deafness, and even muscular atrophy. Recovery usually takes place in two or three months under the influence of a tonic treatment. We need not wonder that this author does not allude to hysterical manifestations in his cases; for at the time he wrote such occurrences among blacksmiths would have been held to be most unlikely. Yet the mode of origin, especially of these paralyses, reminds one forcibly of what we observed in our first case.

One of F. Smith's patients presented a peculiarity worthy of special notice, viz. in addition to marked paresis he had numb and crampy sensations, and was unable to write. This cramp increased when he tried to take a hammer—showing a relationship between professional neurosis and *paralysis by exhaustion*. The last expression appears a convenient one to express this functional akinesis. If "idea" plays a part in its causation, it is an accessory one only, for the idea is born under special physiological conditions. Depressive ideas postulate a temporarily enfeebled state of the organism, just as depressive delirium requires a previously deteriorated physical basis. In melancholia, sad thoughts and their train of delirious pre-occupations arise after other signs of nutritive disturbances have already become manifest. The idea of paralysis can occupy the mind only after the organism has been prepared for it, either by gradual changes or by some sudden mental or other shock. An emotional state is the necessary condition of the subjective representation of powerlessness; it is observed, for instance, in Russell Reynolds' patient, who was exhausted after long walks about London.

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[1] "Paralysis and other Disorders of Motion and Sensation dependent on Idea:" 'Brit. Med. Journ.,' 1869, ii. pp. 378, 835.

[2] 'Lancet,' 1869, i p. 427; 'Brit. Med. Journ.,' 1876, ii. p. 591.

By the suggestion of paralysis to hypnotized or hysterical persons, a painful emotional state is first produced, as shown by the more or less evident revolt of the subject, and it is only when she has become depressed by this emotion that the paresis appears and localises itself. In the same way an opposite, or therapeutical, suggestion acts first by an exaltation of vitality, due to the idea of relief, after which movement begins to be re-established. Moreover, when a paralysis is suggested to even an amenable subject, some resistance is manifested. The assertion that he has lost the use of an arm is not accepted at once; it must be repeated. But if a depressive emotion has been previously called forth, as for instance the announcement of a great misfortune, the suggestion of a local paralysis is much more quickly effectual. Conversely, the previous induction of a happy frame of mind enhances the effect of the therapeutic suggestion, and the resolution of the paralysis occurs much more readily.

The pathogeny of functional troubles due to shock must not therefore be reduced to the mechanism of suggestion. On the contrary, it is possible to explain the effects of suggestion only by a process of shock, which determines, according to circumstances, an erethism or a depression. Mental conditions can be modified only by changes in the nerve-centres, the external causes of which can generally be discovered.

Exhaustion appears to me to be the necessary condition of functional paralyses; and I think this condition may be sufficient to explain the phenomena. The "idea," when it arises, is consecutive only. In many traumatic functional paralyses that appear some time after the shock, idea can thus play an important *rôle;* whilst those which occur at the same instant, before consciousness has returned, are due to exhaustion only.

Clinical Cases.

ON RECOVERY FROM IDIOPATHIC CEREBRO-SPINAL MENINGITIS.

BY J. S. BRISTOWE, M.D., LL.D., F.R.S.

INFLAMMATION of the membranes of the brain is usually and rightly regarded as a very formidable and fatal disease. Yet even in that deadly variety of it, in which the inflammation depends on the presence of tubercles, there is reason to believe that recovery (probably only temporary) occasionally takes place; and there can be no doubt that in the idiopathic affection the prospects of cure, though still gloomy, are sufficiently good to encourage careful and watchful treatment, and to justify hope even in cases that seem to be doing badly.

Of course in cases of suspected meningitis which recover, and where one's diagnosis rests only on symptoms, it is always possible to argue that these have been misinterpreted; and with the more plausibility in most such cases, because the symptoms have for the most part been not only milder, but less distinctive, than they usually are in cases that prove fatal.

I have already, elsewhere, adduced evidence in favour of the possibility of recovey from symptoms of meningeal inflammation due to the presence of tubercles. I propose in my present paper to bring forward cases illustrative of the cure of (or perhaps rather recovery from) meningitis of idiopathic origin.

By a curious coincidence, of which examples are frequently met with in the course of practice, three cases of what I regard as idiopathic, or sporadic, cerebro-spinal meningitis have come under my care in hospital practice during the last eighteen months. Of these, two recovered, and one (the last) died. I add this last case because it tends to confirm the correctness of the diagnosis made in the other two; inasmuch as while it was almost identical with them in symptoms, at the autopsy well-marked meningitis (unassociated with tubercle) was found.

CASE 1.—*Cerebro-spinal meningitis—Recovery.*

M., aged 26, a riveter, was admitted under my care on the 8th of July, 1886. He had been quite well up to the 3rd of the month; on the morning of which day, while dressing, he felt a sharp pain at the back of the head. He went to work as usual; but in a short time was again suddenly seized with violent pain at the back of the head, attended on this occasion with nausea. He tried to go out of the workshop into the open air, but would have fallen had he not been supported; and he vomited. He does not think that he lost consciousness. He was then taken home and put to bed, where he remained until his removal to the hospital. During this time he suffered from almost continuous severe occipital pain, retraction of the head with inability (from pain) to bring it into the natural position, nausea and constipation. There was no history of syphilis, rheumatism, or indeed of any serious illness.

State on admission.—The patient is a slightly-built man, complaining of great pain in the head, which, he says, is now general, and occasionally shoots down the neck, and into the shoulders. The head is strongly retracted and kept rigidly in that position; and there is tenderness on pressure over the spines of the upper two or three cervical vertebræ. There is no affection of the ears. The pupils are not quite equal, but both act well to light and accommodation. There is no oculo-motor paralysis; and the fundi are normal. No trace of paralysis or loss of feeling is present in trunk, face, arms or legs. But the knee-jerks are brisk, and there is slight ankle-clonus. He is quite sensible.

No disease is discoverable in the chest or abdomen. The tongue is coated, the appetite bad, the bowels confined; the urine has a specific gravity of 1030, and is free from albumen; pulse 76, respirations 32. The temperature during the first day varied between 100 and 100·6.

There was no great change in the patient's condition during the next four days. During this time, there was still much retraction of the head, with more or less constant pain in it, and tenderness over the upper cervical vertebræ; his temperature, though tending to fall, and occasionally normal, usually varied between 99 and 100·4; his bowels continued inactive; and his pulse ranged from 54 to 65. But he suffered from occasional paroxysms of very intense pain, confined chiefly to the back of the head, attended with groaning and cries of distress, and lasting from two to three hours or more at a time. These paroxysmal pains at times extended down the neck and arms; and on one occasion were most severe in the sacral region and along the thighs. The attacks usually came on without obvious cause; but once or twice were evidently induced either by attempts to bend the patient's head, or by making him sit up for the purpose of examination. It was observed, also, that there was much tenderness, apparently in the right sterno-mastoid muscle at the level of cricoid cartilage. He did not vomit; he remained quite sensible, and (excepting when suffering from severe pain) slept fairly well. On the 11th, twelve leeches were applied behind his ears.

From the 13th to the 17th there was progressive amendment. He still had occasional attacks of pain, but they were much less severe than they had been; he still complained of tenderness in the cervical spine and right sterno-mastoid, but this was diminishing; and he became able to move his head, so that on the 17th he could bring his chin to within two inches of the sternum. The pulse was still inclined to be slow, and the temperature (which was generally subnormal) ranged from 96·4 to 99. The optic discs were healthy. On the 17th he got up for the first time.

On the 18th, 19th, and 20th, he still on the whole gained ground; but he still suffered from paroxysms of headache, and on the morning of the 20th had an attack of some severity, which lasted for about two hours. His temperature, however, continued subnormal; and the movements of his head were becoming more free. Some tenderness remained in the right sterno-mastoid, and tenderness also was noticed in the left.

With the exception of the occurrence of a not very severe attack of frontal headache early in the morning of the 27th, no incident worthy of notice was observed during the remainder of his stay in the hospital. He gradually lost all trace of headache and of stiffness and pain in the neck, and was discharged apparently well on the 2nd of August. During his stay in the hospital he was treated with iodide of potassium and mercury.

CASE 2.—*Cerebro-spinal meningitis (?).—Recovery.*

George D., a porter, 19 years of age, came under my care on the 23rd of June, 1887. He stated that he had had good health until the previous Easter, since when (although he had had no definite symptoms of illness) he had not felt quite well.

He had for some days been much exposed to the sun, when (on the 19th) he woke up with a severe headache, which (attended with vomiting) continued throughout the day. From that time to the day of admission his headache had not left him; he had been so giddy as to be unable to walk, and occasionally he had seen double. His appetite had been bad, his bowels confined.

On admission.—He was a somewhat spare and delicate-looking youth. His face was flushed, his lips dry. His head was held rather rigidly, and there was pain on movement. The pupils were dilated, but equal, and acted freely to light and accommodation. Neither eye could quite reach the outer canthus; but the left external rectus appeared to be the weaker. He saw double, especially when looking at distant objects or to the left. The optic discs were normal. The tongue, which was thickly coated, was protruded slightly to the left. There was no other paralysis, and no impairment of sensation; the tendon-reflexes were normal, and he was perfectly sensible. The thoracic and abdominal viscera were apparently all healthy.

Pulse 60. Urine, sp. g. 1004, and presenting a trace of albumen. Skin hot and dry. The temperature varied from 101·2 to 101·8.

For the next twelve days the patient's condition underwent but little change. His headache, which was chiefly frontal, continued, and was liable to occasional severe exacerbations; and at times the pain extended down the spine. The head, too, though not retracted, was held rigidly; and he complained of some tenderness in the muscles of the back of the neck. The weakness of the left external rectus continued, with double vision when looking at distant objects or to the left; and nystagmus was observed when the eyes were turned strongly to the right or left. The tongue still pointed to the left when protruded. The arms and legs remained unaffected; but slight ankle-clonus appeared on both sides. His temperature was irregular, and varied from 97·4 to 102·8; each access of severe pain being attended with rise of temperature. He had no recurrence of sickness during this time; his optic discs remained normal; he had no convulsions or delusions, and he retained his intelligence.

During the next month there was, on the whole, improvement. The temperature for the most part was normal or subnormal, and the headache was less constant and less severe. But, at irregular intervals of a few days, the temperature would rise to 100 or 101; and such rises were attended with more or less severe pain in the head, which at times extended down the back, and on several occasions down the left arm to the fingers. Moreover, during this period, he was not unfrequently sick, the sickness occurring chiefly in association with the cephalalgia. The squint, nystagmus and tendency to ankle-clonus continued. But the weakness of the left side of the tongue disappeared; and the stiffness of the neck subsided. His eyes were examined at the end of July by Mr. Nettleship, who reported that the right optic disc was then swollen and hazy; and that, although the left disc was less affected than the other, it was also everywhere more or less hazy.

During the first six days of August, he suffered a good deal from attacks of headache, attended with extension of pain to left shoulder and arm, sickness, and febrile temperature. But after this his improvement was almost uninterrupted. His temperature, with only one or two exceptions, remained normal or subnormal; he lost his headache and (sickness excepting on those one or two occasions) his nystagmus, double vision, and ankle-clonus; his appetite became good; he was no longer confined to bed; and for the most part he felt, and seemed, in good health. The optic discs were re-examined a day or two before he left the hospital; and, although both presented evidences of neuritis, they were manifestly improving.

He went on the 8th of September to a Convalescent Home, where he remained a month, and whence he returned apparently well.

During the first week the patient twice had six leeches applied behind the ears, and a few days later a blister to the nape of the neck. Up to August 5th, he was treated with iodide of potassium and mercury; subsequently to that date, with quinine and iron. On one or two occasions opiates or other sedatives were given to relieve pain.

CASE 3.—*Cerebro-spinal meningitis.—Death.—Autopsy.*

E. P., a boy of 13, was admitted on the 5th of December, 1887. A fortnight previously he had come home from school complaining of pain in the back of the head. From that time he had kept his bed, suffering from headache, loss of appetite, sickness, retraction of the head, and so much general tenderness that he screamed whenever he was moved. He had been delirious and noisy at times. But he had never lost consciousness or suffered from fits.

State on admission.—He was a thin boy, with a pinched expression, and strongly retracted head, complaining of pain in the head and back of the neck, and of pain in the eyes when they were exposed to strong light. He was somewhat stupid and confused, and inclined to ramble in his talk; but, when spoken to with decision, answered questions and did what he was told to do. His eyes were kept closed; the pupils were dilated and equal, and acted to light. There was no obvious oculo-motor palsy. His head and spine and thorax were hyperæsthetic. There was no sign of paralysis anywhere. The skin was harsh and dry; and no tache cérébrale could be obtained. His lips were dry, his tongue thickly coated, and he appeared to have some difficulty in swallowing. The abdominal and thoracic viscera were apparently all healthy. Pulse, 80; respirations, 20; temperature varying between 100 and 101. Urine passed unconsciously. Bowels constipated.

For the next three days his condition remained much the same as on admission. His temperature varied every day from about 99·4 to 100 or 101, and his pulse from 80 to 140. He took little food by the mouth, and seemed to have difficulty in swallowing, but was not sick; and had to be fed mainly by the rectum. The retraction of the head and hyperæsthesia continued; he had delusions at times and was apt to be noisy; he complained of pain in the eyes, and in the back of the head and neck; he passed his evacuations into the bed. It was thought that there was a little weakness of the right external rectus. During the greater part of the time, he lay quietly, but resented interference.

In the course of the 9th, his temperature fell to the normal; and from that time to the 14th inclusive it never rose above 98·4, and was generally subnormal, occasionally falling to 96·4. During this period there was, on the whole, marked improvement; he was still noisy and restless at times, and at times inclined to be drowsy; and his pulse continued variable, and for the most part rapid. But he was sensible, and answered questions correctly; his head, (though still occasionally retracted) assumed for the most part the normal position; his difficulty of swallowing subsided, and although he still objected to food, he was latterly fed wholly by the mouth; and he ceased to pass his evacuations incontinently.

His tongue was still coated; his breath offensive; his urine free from albumen.

On the 15th his temperature rose. In the early morning it was 100·2, and at 9 A.M. 102·2; and it varied between 102 and 102·6 during the rest of the day. In the morning he was quite rational, but was sick and complained of great pain in the head, and had again lost control over his evacuations. His pulse was rapid (154); his pupils were dilated and equal; there was no strabismus. No retraction of the head was observed early in the day, but it came on strongly later.

During the greater part of the 16th, the temperature varied from 100·2 to 101·4: but at 8 P.M. it had fallen to 99·8. He had been very noisy during the night, but became drowsy and difficult to rouse in the course of the day; and, when roused, said that he felt worse and had pains all over him. He took food well, but vomited two or three times. His tongue had again become dry and brown, and his lips covered with sordes. Pupils normal. Pulse 124.

From the 17th to the 21st inclusive the temperature again was either normal or subnormal; and again there was marked improvement. He was very drowsy for the first day or two; but he became perfectly intelligent, answering questions readily; he lost his cephalalgia, retraction of head and sickness; he took food fairly well; and regained control over his evacuations. His pulse was usually about 120. On the 21st he complained of pain in one of his feet.

From the 22nd to the 27th there was again some elevation of temperature, which varied for the most part between 99·4 and 100·8, but on the 24th rose to 102·2. During this time, his symptoms varied somewhat; but he was evidently not so well as he had been. He complained at times of frontal headache, and at times was free from pain; his head was occasionally strongly retracted, but on the whole was freely movable; he was sick once or twice; he was generally irritable, and disinclined to answer questions or to take food. His tongue was inclined to be dry; his pulse was generally over 120; for the most part he had control over his evacuations. He had never previously allowed his eyes to be tested ophthalmologically, but now they were carefully examined on two occasions, and the fundi were found to be perfectly normal.

From this time until the 10th of January there were no very great changes in his symptoms, excepting that his emaciation and weakness, which had been progressively increasing since admission, became extreme, and he passed into a condition of apathy, in which he seemed to know and occasionally to observe what was going on, but for the most part vouchsafed no reply to questions save an occasional nod, and practically refused all nourishment. He looked intelligent; did not appear to suffer any pain in the head or elsewhere, although when he was moved or washed he would cry out and resent what was being done; lay for the most part on his back without any retraction of the head; and passed his evacuations without any attempt to control them. He had to be fed through the nose, and was occasionally sick. He suffered from no attacks of unconsciousness or fits; and no signs of paralysis were detected. It may be added that his tendon-reflexes were normal. During all this time his temperature fluctuated: for a day or two being normal or subnormal; and then for a day or two varying between 99 and 101, or a little over. His pulse was generally quick, and on one occasion was counted 160 in the minute. Although in many respects the symptoms, and more especially those which seemed to be directly due to cerebro-spinal disease, appeared latterly to have undergone decided amelioration, it was quite clear that the patient was slowly and surely sinking from sheer debility.

The treatment consisted mainly in the occasional exhibition of calomel purges, and application of leeches or blisters to the temples or behind the ears; together with the employment of iodide of potassium and mercury during the first ten days, and of quinine during the next fourteen days, and again of iodide of potassium and mercury during the last eight or nine days.

During the greater part of Jan. 11th no change was observed. At 6 P.M. his temperature was found to be 103·2; and a quarter of an hour later he had a convulsive fit, which lasted about two minutes. He never regained consciousness. At 8 P.M. his temperature was 100·4; between 8 and 9 he had two more slight convulsive attacks. At 11 P.M. his temperature was 106·2. He died at 3.30 the following morning. His temperature immediately after death was 101·6.

Autopsy.—The corpse was extremely emaciated. All the thoracic and abdominal viscera were perfectly healthy, and free from tubercles. On opening the cranial cavity, the surface of the brain was seen to be dry, and the convolutions flattened. There was an abundant exudation of soft opaque lymph covering the under surface of the pons varolii, medulla oblongata, and cerebellum, and extending forwards so as slightly to involve the optic commissure. But there was no inflammatory exudation, either in the anterior or middle fossæ, or along the sylvian fissures. The lateral ventricles contained four to five ounces of serum, and the commissures were softened. But generally the substance of the encephalon was healthy. There were no tubercles, no disease of bone, no affection of venous sinuses or of arteries. There were slight traces of inflammation of the membranes of the upper part of the cord. But the cord itself and the vertebral column were healthy.

It will be readily admitted, I think, that the symptoms in all these cases were such as are apt to attend, and are fairly distinctive of, meningitis of the base of the brain and upper part of the cord. They all presented headache (not necessarily limited to the back of the head); retraction of the head, or rigidity of the neck, with pain and tenderness at the back of the neck; pain shooting down the spine and into the limbs or general hyperæsthesia; and remittent febrile attacks, usually attended with aggravation of headache and sickness.

But the symptoms of meningitis are always very variable; and it is not surprising, therefore, that some of the symptoms frequently met with were absent from one or other of the cases, or but slightly marked, and that consequently, as to details, they differed a great deal among themselves.

None of them presented any paralysis of limbs; but in the first two slight ankle-clonus was observed temporarily. In the last it was not detected; but this may have been due to the fact, that the patient always complained of pain when the feet were handled, and thus prevented examination.

In the second case, there was paralysis of one of the external recti with double vision; in the third, at one time it was thought that one of the same muscles acted imperfectly; in the first, slight inequality of pupils was noted. But with these exceptions there was no oculo-motor paralysis. For the most part the fundi of the eyes were healthy. In the first case, no optic neuritis was discovered. In the second case (which was under treatment for eleven weeks), slight optic neuritis became apparent at the end of five or six weeks, but had nearly subsided at the time the patient left the hospital. In the third case, the eyes were examined four or five weeks from the onset of the boy's illness, and no optic neuritis was then found. No subsequent examination could be made; so that whether or not it supervened later I cannot say. It is noteworthy that, in the case in which there was an obvious and persistent squint, there was also slight nystagmus. In the first two cases the pulse, though variable, tended to be slow; in the other the pulse was also variable, but usually very rapid. In the last case, although the specific symptoms of meningitis were not more pronounced than in the other two, the illness from the beginning appeared of a graver type; and it is not surprising, therefore, that in this case only were there mental disturbance and loss of control over the evacuations. It is interesting, too, that in this case the specific cerebral symptoms had almost completely subsided for some little time before death; and that this event appeared to be brought about mainly by progressive innutrition and debility. The peculiar apathetic and passively obstinate frame of mind, which characterised this boy during the last week or two of life, is a curious and interesting feature of some fatal cases of chronic affection of the membranes or surface of the brain.

I am afraid that no definite rules of treatment can be derived from my cases. I believe that leeches and counter-irritants to the temples or behind the ears, and opiates or other sedatives for the relief of pain or restlessness, are often of much value in such cases, and were useful in these. And I am inclined to think that iodide of potassium and mercury are serviceable during the earlier stages, and quinine at a later period, especially if there be signs of amendment. But, on the whole, I suspect that judicious nursing and attention to small and various details is of more real value than drugs.

ABSENCE OF MOTOR AREAS IN THE BRAIN OF AN EPILEPTIC.

BY J. MUNRO SMITH, L.R.C.P. (LOND.), M.R.C.S.,

Lecturer on Physiology, Bristol Medical School; Demonstrator of Morbid Anatomy, Bristol Royal Infirmary.

A. B., aged 51, an epileptic, fell into the fire whilst in a fit and received severe burns, from which he shortly afterwards died.

The following history was obtained from his brother. As a boy he was of average intelligence, and learnt to read and write without difficulty. In his eighth year he became subject to general convulsions, attended by loss of consciousness, each fit lasting a few minutes. When twelve years old his left foot began to turn in, and as he grew up he had well-marked talipes equino-varus. The left arm about the same time showed a tendency to flexion, and ultimately the flexors of the fore-arm and hand became firmly contracted. The other muscles were in working order; he could walk, and had good movement in the muscles of the thigh and calf. He never followed any definite employment, being slow to understand and lazy; he read the papers and did work that he liked, but was idle and useless. The fits occurred with great regularity once a day during the last twenty years of his life, usually at night: this was noticed by his brother, who slept in the same room with him during that period. Latterly he had occasionally suffered from attacks resembling "petit mal." He had never any unilateral convulsions, so far as could be ascertained.

At the *autopsy*, the body was well nourished and of average dimensions: the left arm and foot showed the deformities above described. Rigor mortis was well marked.

On removing the vault of the skull a cyst was observed on the right hemisphere, under the dura mater, with which it had only very slight connections. It was situated over the fissure of Rolando; was about the size of a Tangerine orange, somewhat oval in shape, and full of clear cerebro-spinal fluid. The walls were composed of delicate connective tissue arranged in layers, but thin enough to be translucent.

The upper part of the fissure of Rolando was not encroached upon, and is seen in the drawing at R. The superior, middle, and inferior frontal convolutions were intact. The supramarginal and angular convolutions were small, firm, and flattened; the latter (B) was corrugated and irregular. The ascending parietal and ascending frontal convolutions were apparently absent; the remains of them being pressed down, flattened, and atrophied, forming the smooth floor of the cavity (c c). The walls and floor of this cavity were almost devoid of grey matter.[1] On the under surface of the hemisphere the right temporo-sphenoidal lobes were much smaller than on the opposite side. The horizontal limb of the fissure of Sylvius was apparently continuous (as is sometimes the case in healthy brains) with the interparietal sulcus. All the atrophied parts were of firmer consistence than is normal.

On making sections of the brain, the basal ganglia appeared to be the same on both sides; this was the case, too, with the internal capsule. The tegmentum of the right crus was a trifle smaller than the left, but microscopic sections (not very successful ones) showed no marked degeneration.

Most unfortunately the spinal cord was not kept. No change could be detected in the medulla.

This case seems to me to be of interest in its etiology and symptoms.

As to causation: the history is not consistent altogether with the disease being congenital, or the result of hæmorrhage; post-mortem appearances were also against the latter, and the lesion was not due to hydatids probably. Perhaps some localised meningitis or polio-myelitis led to an atrophy of the cortex, which slowly increased, the shrivelled brain being replaced by cerebro-spinal fluid; or possibly there was blocking of an artery, with consequent malnutrition and degeneration. No thrombosis, however, was found. There was no history or sign of any injury to the skull. In its clinical aspects, also, I think the case worthy of brief record, as showing the amount of atrophy of grey matter that may occur without corresponding loss of function. Had the regions involved been *suddenly* affected, the paralysis would doubtless have been very extensive, for the motor centres for the face, arms, legs, and partly for the trunk, were wasted; yet there was no facial paralysis, and all the muscles of the trunk, thigh, and leg were in good condition both as to use and dimensions. This may be accounted for, probably, by the extremely slow nature of the change, so that compensation occurred; and the gradually diminishing function of the parts affected was assumed by neighbouring convolutions, such as the marginal gyrus, which has been shown by Schäfer and Horsley to contain special centres for the elbow and shoulders, trunk muscles, thighs, digits, &c. In this respect this brain may be compared with one figured in the 'Medical

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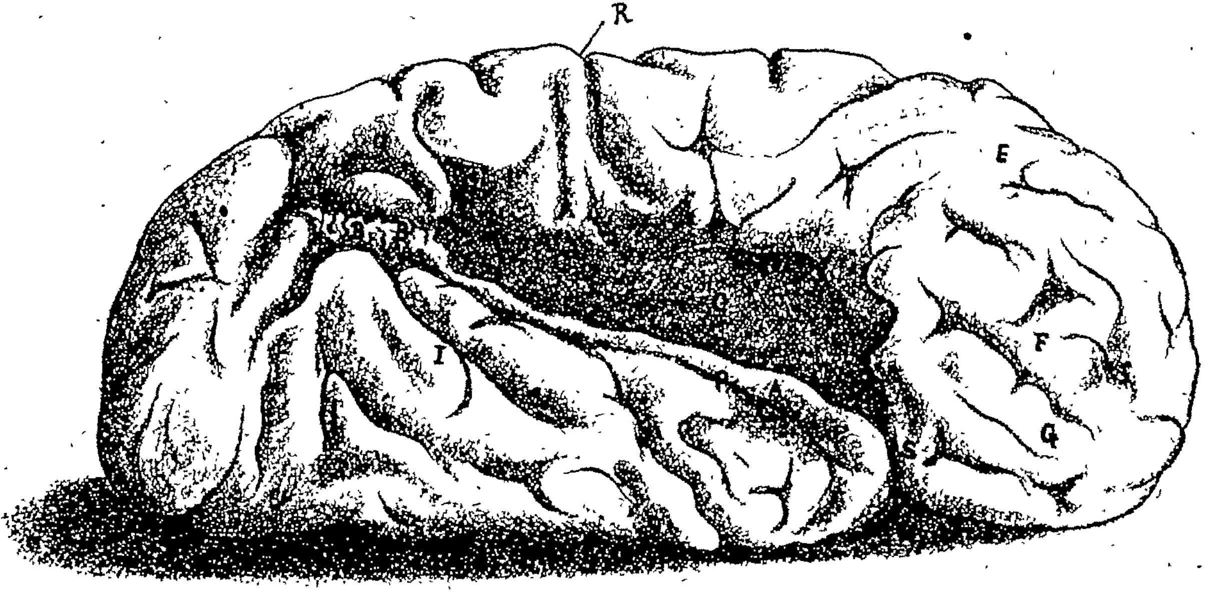
[1] The various sulci on the surface of the brain were so altered that accurate mapping out of the parts involved was difficult. I am indebted to Dr. Shingleton Smith, Dr. Michell Clarke, and Mr. B. Baskett for help in this.



G M Smith, lith.

Danielsson & Co, imp

Journal' of April 21st, by Dr. Byrom Bramwell, in which a large sarcomatous tumour occupied the motor region of the right cerebral cortex, and was not associated during life with any paralysis whatever. It is true that in the present case there was permanent contraction of the foot and arm, but comparatively very few muscles were affected. The want of atrophy of the internal capsule of the corpus striatum also bears out the idea, that increased function in one region had counterbalanced increased loss in another.

DESCRIPTION OF PLATE.

A. Atrophied supra-marginal convolution.
B. Angular ditto.
C. C. Cavity.
E.
F. } First, second, and third frontal convolutions.
G.
I. Temporo-sphenoidal convolutions.
P. First temporo-sphenoidal fissure.
R. Fissure of Rolando.
S. Commencement of Sylvian fissure.

CASE OF PRIMARY ATHETOSIS.

BY ALEXANDER M. MACALDOWIE, M.D., F.R.S.E.

THE following case is of sufficient rarity to merit publication.

Mrs. P——, aged 62, March 26, 1887. Patient states that she has always been of an excitable and "nervous" disposition, but has never had any fits, chorea, nor attacks similar to the present. No family history of nerve disease nor of syphilis nor rheumatism can be elicited.

For several weeks previous to the present illness her friends have noticed slight twitchings in the left hand, but the patient has been able to perform her customary house duties. The attacks began ten days ago with pain on the right parietal region. A few days afterwards convulsive movements began in the left hand and arm and have continued till now. The face, trunk, or other limbs have not been affected. The husband states that the movements, as a rule, cease during sleep, but occasionally slight twitchings occur, generally towards morning. The general health is good; the patient eats and sleeps fairly well, but feels exhausted from the continual movements.

Patient is a fairly nourished, healthy-looking woman. Complexion fair. The left hand, fore-arm, and arm are the seat of slow rhythmical movements affecting chiefly the extensor muscles. These usually occur in cycles. At the beginning of the cycle the hand rests on the lap with the fingers semiflexed and drawn together, the wrist is dropped, and the elbow semiflexed. The three inner fingers then extend and separate by slow rhythmical movements till a position of hyperextension is attained. The hand is then drawn backwards and towards the ulnar side in the same manner, and the movements gradually spread up the extensors of the fore-arm and arm until the limb is stretched almost horizontally behind the patient as she sits in the chair, with all the joints, except those of the thumb and index finger, fully extended, the three inner fingers widely separated, and the palm directed upwards, backwards, and inwards. After remaining in that position for ten to fifteen seconds, during which the slow rhythmic movements continue, the cycle is completed by a series of movements almost exactly the converse of the preceding, till the hand regains its position on the lap. The thumb and index finger are scarcely, if at all, affected, but remain slightly flexed and pressed together. These cyclic movements occur with great regularity when the patient's attention is withdrawn, during conversation, &c., but she is able, by an effort of will, to check them at any period. She is, however, unable to prevent the fingers from performing the peculiar movements and contortions characteristic of athetosis. These irregular movements are aggravated by any attempt at voluntary movement. There is no pain connected with the muscular contractions; the patient complains only of numbness in the hand and arm. There is also a slight loss of sensation, but no coldness nor lividity, nor loss of power in the affected limb. Deep reflexes are obtained on percussion over the ulnar side of the fore-arm. The superficial reflexes are markedly increased. Tickling the skin in front of the fore-arm produces tonic contraction of the portion of the flexor muscles supplying the three inner fingers, which are shut and pressed firmly against the palm. Tickling the skin on the back of the fore-arm, on the contrary, appears to affect the extensor minimi digiti alone; the little finger standing rigidly out from the rest in a state of hyperextension. The electrical excitability of the affected muscles is increased. The knee-jerk appears exaggerated on both sides. The patient suffers from constant pain over the right parietal region, but it is not so severe as at the beginning of the illness. There is tenderness or percussion over that region. The eyesight is normal, ophthalmoscopic appearances natural.

The patient was treated at first with bromide of potassium, afterwards with arsenic, and blisters were applied behind the right ear. The movements gradually ceased, and she was quite well in three weeks. She has had no relapse of the symptoms since. (June 1888.)

The chief point of interest in this case lies in the fact, that the disease was primary or idiopathic, and that it terminated in complete recovery. Our knowledge of the pathology of this affection is mainly hypothetical, but the majority of authorities are of opinion that one or other of the central ganglia is the site of the lesion. The clinical history of this case, however, especially the pain and tenderness over the parietal region, points to some coarse disease of the cerebral cortex of an irritative character as the initial lesion. The right ascending frontal and ascending parietal convolutions were doubtless the parts of the cortex affected. The limitation of the movements to the three inner fingers is a peculiarly interesting feature in this case, as in the majority of cases recorded the thumb and index finger are the digits particularly affected with spasms.

MUSCULAR ATROPHY OF THE PERONEAL TYPE AFFECTING MANY MEMBERS OF A FAMILY.

BY W. P. HERRINGHAM, M.B.

Two years ago Dr. Tooth described, in a thesis for the M.D. of Cambridge,[1] a new type of progressive muscular atrophy, beginning in the legs and showing a hereditary tendency. It had been independently distinguished, a short while before, by Professor Charcot and M. Marie. For the last year I have had under my care, at the West London Hospital, a remarkable case of the disease.

Charles Wheeler, aged 22, a fine-looking young man, by trade a holder-up for riveters, was sent to me at the West London Hospital by Dr. Seymour Taylor, for electrical treatment. It is now five years since he noticed a weakness in his legs, the right being considerably the earliest affected. It has caused a deformity in his feet, for which he once underwent tenotomy of the right tendo Achillis, and which leads him to wear out the toes of his boots; he suffers much from cramp in the legs; for fifteen months he has noticed weakness and wasting in the left hand, and for nine months in the right.

The feet are much arched, the instep being raised, and the length of the foot proportionately diminished. On the right side the first phalanx of the big toe is bent upwards, the second downwards; the same upward bend is less observable in the first phalanx of the other toes. The heels are slightly drawn upward, by contraction of the tendo Achillis.

There is great wasting of the muscles in the tibial and peroneal regions, and some, which is increasing, in the calf. The muscles in front of the thigh have also begun to waste.

Since the foot cannot be properly bent upwards owing to the weakness of the tibial muscles, the knees have to be raised higher than usual to keep the toes off the ground, and when the foot is lowered the ball of the toes touches the ground first. At the same time, since the thigh muscles are not early affected, the foot points straight forward as in health, and there is no irregular throwing of the feet. I have never seen a case of infantile paralysis affecting both legs where there was not some paralysis of one or both thighs, and this causes the foot to be turned out in a way quite different from the gait I am describing. I should think therefore that in a young person, in whom alcoholic paralysis is not likely, this way of walking might usually be ascribed to this disease. I have seen two or three cases in the streets since I have been on the look-out for it. The toes of the boots are of course worn out quickly, just as in spastic paraplegia.

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[1] See 'BRAIN,' Vol. X. 1887, p. 243.

The look of the hands was just that of the usual form of progressive muscular atrophy, the thenar, hypothenar, and interosseous muscles being wasted. Since he has been under me the forearm muscles, both flexor and extensor, have wasted on both sides. The extensors of the thumb were the first muscles above the wrist to waste.

Fibrillar tremors have been very marked throughout, and have been seen in muscles which did not perceptibly waste till later.

I could not find any anæsthesia to touch or temperature, and except for the cramps he has had no pain.

When he first came, faradic contractility was diminished in the wasted muscles; but there was no R D to galvanism. The muscles contracted more easily to stimulation of the nerve than when they themselves were touched. During this year R D has appeared in several muscles, both of arm and leg.

The knee jerks were normal at first, but are now diminished.

I took a tracing of the tremor, and found it to be at the rate of ten or twelve per second, that of normal muscular contraction.

He brought me a cousin—George Milton, 29, also a big strong man. He was quite healthy until five years ago, when his legs and feet began to grow weak. He has noticed weakness in his hands for two years. He presented the same appearance of the legs, though in a less degree. In the hands the thenar, hypothenar and abductor indicis muscles are slightly wasted. There was tremor of the calf, and no trace of anæsthesia. I have not had the opportunity of testing the muscles electrically.

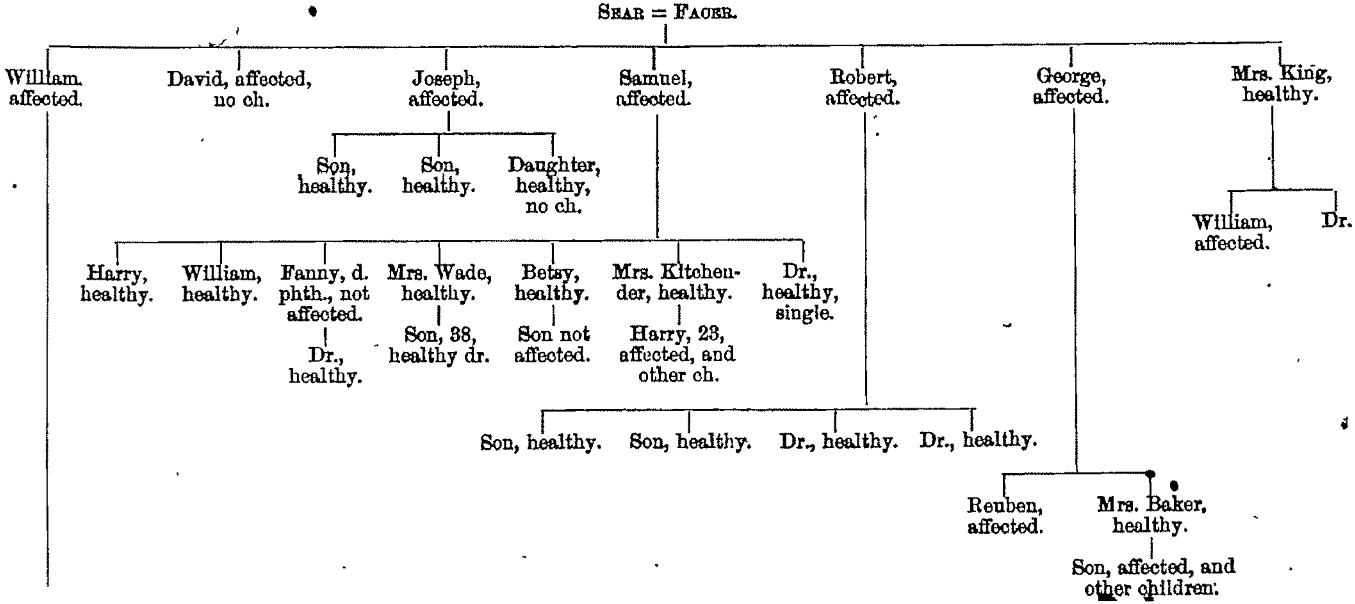
I could not in either case find any hypertrophy of muscles, and with the exception of syphilis in the first patient both had been perfectly healthy till the onset of the present affection. They now enjoy good health in every other respect, and the viscera are quite sound.

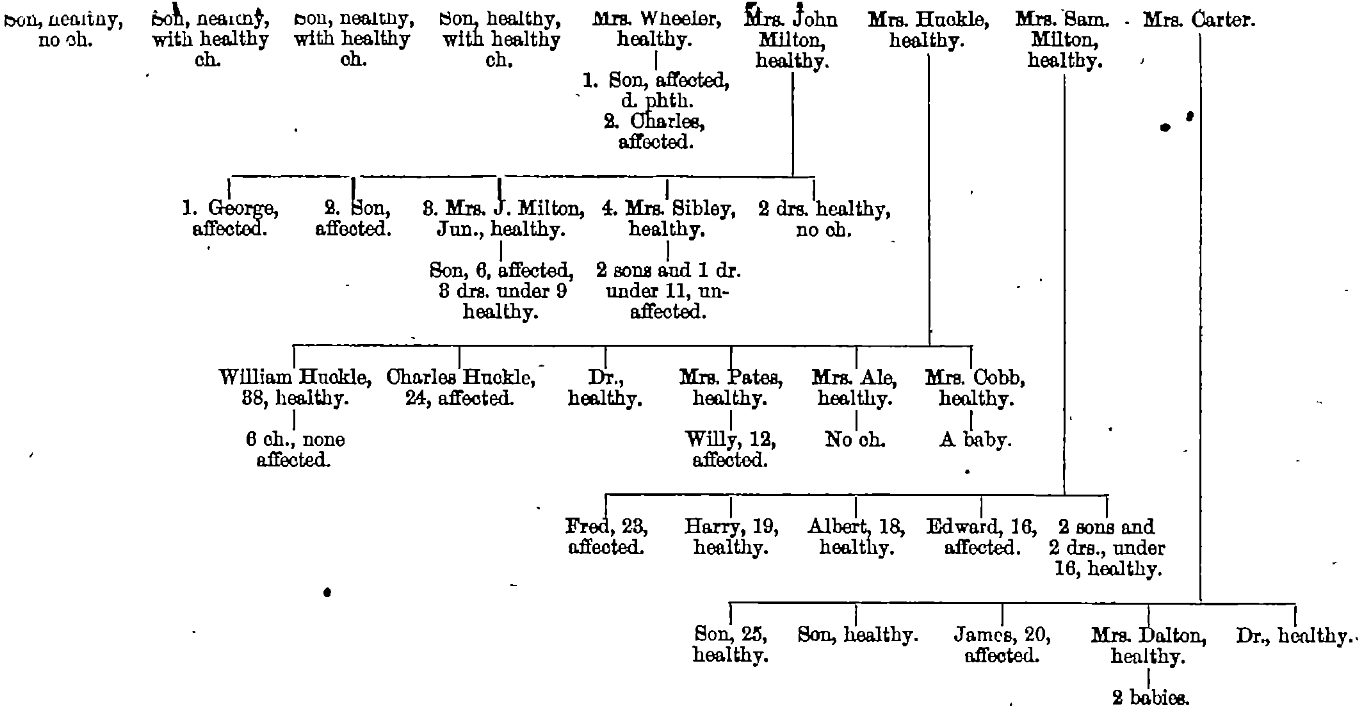
The disease is quite familiar to them, for, as will be seen, many males of the family are similarly affected; but they do not usually seek medical treatment for it. On going into the country to see the relations, I could not find that any of those whom I saw had ever been under a doctor for the complaint.

The family history can be traced through five generations, and is as follows:—

Sear married Facer, and had seven children; six sons and one daughter:

1. William S., affected in legs and arms, died at 70, of apoplexy.
2. Joseph S., affected.
3. David S., slightly affected in legs.
4. Samuel S., affected, died at 65.


Shar = Facer.
William. affected.
David, affected, no ch.
Joseph, affected.
Samuel, affected.
Robert, affected.
George, affected.
Mrs. King, healthy.
Son, healthy.
Son, healthy.
Daughter, healthy, no ch.
William, affected.
Dr.
Harry, healthy.
William, healthy.
Fanny, d. phth., not affected.
Dr., healthy.
Mrs. Wade, healthy.
Son, 38, healthy dr.
Betsy, healthy.
Son not affected.
Mrs. Kitchender, healthy.
Harry, 23, affected, and other ch.
Dr., healthy, single.
Son, healthy.
Son, healthy.
Dr., healthy.
Dr., healthy.
Reuben, affected.
Mrs. Baker, healthy.
Son, affected, and other children.


Son, healthy, no ch.
Son, healthy, with healthy ch.
Son, healthy, with healthy ch.
Son, healthy, with healthy ch.
Mrs. Wheeler, healthy.
1. Son, affected, d. phth.
2. Charles, affected.
Mrs. John Milton, healthy.
Mrs. Huckle, healthy.
Mrs. Sam. Milton, healthy.
Mrs. Carter.
1. George, affected.
2. Son, affected.
3. Mrs. J. Milton, Jun., healthy.
Son, 6, affected, 3 drs. under 9 healthy.
4. Mrs. Sibley, healthy.
2 sons and 1 dr. under 11, unaffected.
2 drs. healthy, no ch.
William Huckle, 38, healthy.
6 ch., none affected.
Charles Huckle, 24, affected.
Dr., healthy.
Mrs. Pates, healthy.
Willy, 12, affected.
Mrs. Ale, healthy.
No ch.
Mrs. Cobb, healthy.
A baby.
Fred, 23, affected.
Harry, 19, healthy.
Albert, 18, healthy.
Edward, 16, affected.
2 sons and 2 drs., under 16, healthy.
Son, 25, healthy.
Son, healthy.
James, 20, affected.
Mrs. Dalton, healthy.
2 babies.
Dr., healthy.

5. Robert S., affected.
6. George S., affected, died at 74.
7. Mrs. King, healthy.

1. William S. had nine children; four sons and five daughters. The four sons were all quite healthy; three of them are married. One lives at home; his children are healthy.

Two others are in America, and correspond frequently. They have never said that anything of this sort has occurred in their children.

The five daughters were all healthy and all married:

1*a*. Mrs. Wheeler had two sons; an elder died at 19 of phthisis. He was a shorthand clerk, and had before death become affected in the hands so that he could not write comfortably. He died ten years ago. The state of his legs is doubtful.

The next son is Charles, the patient.

1*b*. Mrs. John Milton has had two sons and four daughters.

The eldest son is George, the patient.

The second son has written lately to say that his feet are getting bad. He is about 20 years old.

The daughters are all healthy. Three are married, viz.:

Mrs. John Milton, jun., has one son, aged 6, who is "weak on the foot; not right in the feet," and three daughters under 9, who are healthy.

Mrs. Sibley has two sons under 11, healthy, and one daughter, who has abscesses in the legs.

Mrs. Page, no children.

1*c*. Mrs. Huckle has two sons and four daughters:

William, 38, healthy, is married, has two sons and four daughters. The eldest son is about 13, and both are unaffected.

Charles, 24, began to waste in the legs at 10. The left leg is the worst; it is extremely wasted below the knee, both in the calf and front of the leg. The foot is a hollow claw, the arch being so high and narrow that the tarsus projects on the dorsum like an abnormal swelling; it is also slightly curved inwards. There is some wasting and tremor of both thenar muscles. He says his hands are getting weak, and that he cannot hold things steady. There is no pain or anæsthesia.

The daughters are all healthy. Three are married:

Mrs. Pates has five children. Willy, aged 12, the eldest boy, walks badly, and is considered affected.

Mrs. Cobb has only a baby.

Mrs. Ale has no children.

1*d*. Mrs. Sam Milton has six sons and two daughters:

Fred, 23, wasting of legs, feet arched, talipes equinus slight, and tremors in legs and hands; no pain or anæsthesia. He thinks the disease began two years ago.

Harry, 19, and Albert, 18, healthy.

Edward, 16, legs much wasted, feet much arched, tremors in legs and hands; no pain or anæsthesia. He has walked badly since he was 6.

Arthur, 6, and Herbert, 3, are healthy.

The two daughters, both under 15, are healthy.

1*e*. Mrs. Carter, now dead, had three sons and two daughters:

The two eldest sons, of whom the elder is only 25, are healthy.

James, aged 20, is affected.

The daughters are quite healthy. One, Mrs. Dalton, has two babies.

None therefore of William Sear's children are affected; but of fifteen daughters' sons, eight are already affected, two are under 20, and two more are under 6 years of age.

2. Joseph Sear had two sons, who were healthy, and one daughter who had no children.

3. David Sear had no children.

4. Samuel Sear had two sons, and five daughters. The sons were both healthy. The daughters were also all healthy; four were married.

4*a*. Fanny, died of phthisis, had one daughter, who was healthy.

4*b*. Jane, Mrs. Wade, had one son, now 38, and one daughter, both healthy.

4*c*. Betsy, had one son, healthy in limbs, but weak in the eyes.

4*d*. Eliza, Mrs. Kitchender, has children; the eldest son Harry, now 23, is affected.

5. Robert Sear had two sons and two daughters; they were healthy, but nothing has been seen of them lately.

6. George Sear had one son and one daughter.

6*a*. The son, Reuben, is very badly affected.

6*b*. The daughter, Mrs. Baker, herself healthy, has a family of sons and daughters. The eldest son, now in America, became affected in the legs.

7. Mrs. King (born Sear) had one son and one daughter.

7*a*. The son, William, now dead, was badly affected.

I can discover nothing about the Facers, except that Mrs. Sear (born Facer), the mother of this tree is said by her grandchildren to have been crippled in her legs.

There are a few points on which I will add remarks.

1. These cases do not give any corroboration to the opinion that the disease is a neuritis.

2. This, the daughter's son inheritance, is peculiar to this family, not to the disease.

3. This form makes one wonder what inheritance is. That the diseased tissues of a consumptive father should be so represented in his spermatozoon as to cause his child to fall

into consumption is remarkable enough. But that the women of this family, themselves even uncommonly buxom and healthy, should be able to select their children, and transmit to the males alone tissues unlike their own, and endowed with a regular form of weakness which they do not themselves possess, is still more marvellous. It seems as if the daughter of a diseased father carried from the beginning of her life ova of two sexes, the female healthy, the male containing within it the representation of the father's disease.

4. Although in these cases both peroneal and tibial muscles are alike atrophied, so that it cannot now be said for certain which were first affected, yet there is in several a slight lifting of the inner border of the foot, and a slight increase of the concavity to the inner side, which seem to show that the peronei were at one time the most diseased. The talipes equinus is due to the unopposed contraction of the muscles at the back of the leg, at a time when those at the front were alone affected.

PERIPHERAL NEURITIS IN ENTERIC FEVER.

BY HENRY HANDFORD, M.D., M.R.C.P.,

Physician to the Nottingham General Hospital.

PARALYSIS is an occasional sequel of enteric fever. Nothnagel, in 1872, made this the subject of a special memoir; and, mentioning among other forms strabismus, paralysis of the portio dura, motor paralysis of individual spinal nerves, such as the ulnar or peroneal, and local anæsthesia, suggested that the cause of these was similar to that of diphtheritic paralysis.

Murchison[1] says, "Now and then these attacks of paralysis, particularly in the legs, terminate in atrophy of certain of the muscles, and I have met with several instances where permanent distortion has resulted. One of my patients, a girl aged 18, had all the signs of paralysis of the right third nerve, in a marked degree, throughout an attack of enteric fever this had first occurred fourteen years before, after an attack of measles, but for many years had almost disappeared until the seizure with enteric fever, on convalescence from which only slight ptosis remained."

Gowers,[2] in speaking of inflammation of nerves, says, "Acute diseases may also lead to neuritis. Small-pox, typhoid fever, and others may cause simple neuritis."

We may take the occurrence of neuritis during or after enteric fever as unquestioned, but, as the number of recorded cases is not yet very large, I venture to add the following three somewhat at length. They are of some interest from the fact, that they were all three very similar in their course, and were all strictly limited to the lower part of the ulnar nerve. I have given the cases with considerable detail, in the hope of throwing some light upon the cause of the neuritis.

Ziegler,[3] speaking of neuritis, says, "Other forms occur in which the irritant inducing the inflammation is brought to the nerve directly by way of the blood or lymph. These irritants are, so far as we know, chiefly of an infectious nature: thus in typhus, small-pox, typhoid, and diphtheria, we meet with simple or multiple neuritis, which we can only regard as direct results of the general infection."

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[1] 'Continued Fevers,' 1884, p. 568.

[2] 'Diseases of the Nervous System,' 1886, p. 53.

[3] 'Special Pathological Anatomy,' vol. ii. p. 843.

Gowers,[1] on the contrary, attributes some of such cases to pressure or over-extension, combined with impaired health. He says, speaking of diseases of the ulnar nerve: "It is probable that pressure often acts by setting up neuritis. . . . It is much more common for the nerve to suffer in long-continued flexion of the elbow, without external pressure; and from this cause the nerve is sometimes paralysed during sleep. I have three times seen a sleep-palsy of the ulnar nerve. The tension may set up neuritis, and is especially effective if the resistance of the tissues to morbid influences is lessened by general ill-health. Many persons must have noticed that if, when out of health, they sleep with the elbow bent, they wake with tingling, and even loss of feeling, in the region supplied by the ulnar nerve, although this may not happen when they are in good health. If the ill-health is profound, as in an acute illness, local neuritis of considerable intensity may be set up. Thus, a lady, a few days after childbirth, who was prostrated by a long, exhausting labour, noticed tingling in the side of the hand, and, whenever she bent her elbow, a sensation 'as if she had knocked her funny bone.' Paralysis of the muscles supplied by the ulnar nerve followed in a few hours, and in a fortnight there was distinct wasting. When I saw her, six months later, the ulnar nerve behind the elbow was distinctly thickened. She had a similar and more transient attack after a previous confinement. *Another patient had a similar palsy during the course of typhoid fever. Bernhardt has described a case of palsy of both ulnar nerves during the course of typhoid.* In such conditions, not only is the tissue health lowered, but warning sensations are often unperceived."

Having now by the somewhat, perhaps, too copious quotations explained the divergence of opinion as to the intimate cause of neuritis in typhoid fever, I will leave the further discussion till after the narration of the cases.

CASE I.—The first case relates to H. M., a lace-maker, aged 21, who was admitted under my care into the Fever Ward of the Nottingham General Hospital on Feb. 23rd, 1887, suffering from enteric fever. His illness had begun a fortnight before admission with severe diarrhœa and vomiting, but he only took to his bed the day before coming in. He had a very severe attack, the temperature being frequently between 104° and 105° for days together. There were much diarrhœa, and six free hæmorrhages, besides albuminuria to a moderate degree. Sore throat existed at the time of his admission, the tonsils and the mucous membrane of the fauces being congested and of a claret colour; later a few shallow ulcers appeared, affecting also the gums and tongue; but there never was any membrane or any resemblance to diphtheria. Subsequently, after the throat had been well for a week or more, he suffered from laryngitis, which, however, did not cause any difficulty in breathing, but revealed itself only by huskiness and eventually by complete aphonia. He was too severely ill for a laryngoscopic examination to be satisfactorily carried out. Towards the latter part of his illness he suffered from dry pleurisy of the left side, with a distinct friction rub, lasting for several days. During most of his illness he took from ℥ij to ℥vj of brandy in the twenty-four hours, and for days as much as ℥x. The duration of the pyrexia was known to be thirty-four days, and, probably, at least fourteen more: and in addition there was an evening rise for six days further. There was no stiffness of the limbs or muscular trembling, such as is not uncommonly met with in severe cases of enteric fever.

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[1] Loc cit., p. 75.

On March 23rd he first complained of "numbness" of the little and the ulnar side of the ring fingers, and of the ulnar border of the hand. On carefully testing the common sensibility, it was found to be greatly diminished over the area mentioned. There was at first some tenderness on pressure on the hypothenar muscular mass. In this case no tenderness was elicited along the course of the ulnar nerve. The affected fingers could not be abducted at all, nor completely extended; but remained with the second and third phalanges partially flexed. This condition of extension of the first joint and flexion of the second and third is, of course, due to paralysis of the interossei and lumbricals. The two outer lumbricals being supplied by the median, and therefore unaffected, prevented this position being assumed by the index and middle fingers. The affected fingers could be flexed completely, showing that the long flexors were uninjured. The movements of the thumb were also impaired, especially adduction and flexion, and the movement of approximating the tip of the thumb to the root of the little finger. The opponens and abductor were unaffected. There was evident wasting, affecting the muscular masses of the hypothenar eminence, and of the first interosseous space. On grasping the affected muscles they were found to be soft and flabby. The contrast with those of the other side was distinct, but became more marked later on as the muscles of the unaffected hand recovered their normal tone and mass after the wasting due to the fever. The next figure shows that the ulnar margin of the affected hand is almost a straight line instead of being convex, as in a well-developed muscular hand.

On April 11th the note in the case-book runs:—Patient is improving, but the feeling does not return to the left little finger and side of the hand. Loss of sensation extends as far as the head of the ulna. There is also now most distinct difference in temperature. The affected parts are cold. The ulnar side of the ring finger is distinctly colder than the other side, which is of the same temperature as the rest of the hand. There is also a subjective sensation of cold.

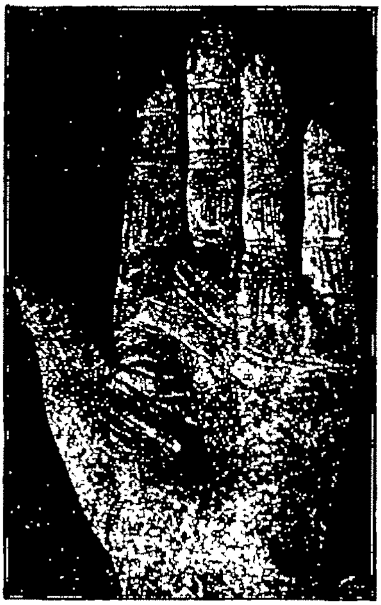

FIG. 1.—Taken directly from a photograph from an "untouched" negative. Notice the straight ulnar border of the hand, and the separation of the little from the ring finger.

April 14th.—Patient has been washing windows: the affected fingers are very cold and quite red, the rest of the hand being unaltered in colour.

May 9th.—The skin over the affected area is becoming *thin, wrinkled, and glossy.*

June 2nd.—Patient returned from a convalescent home much improved in health and strength, but the condition of the hand was not appreciably altered. A complete electrical examination was now carried out for the first time, as no suitable battery was available in the Fever House. The faradic current was derived from two quart Leclanché cells, and the secondary coil of a Du Bois Reymond sledge was 7 cm. distant from completely covering the primary. The current was stronger than could be endured on the sound hand. No response whatever could be elicited from the abductor minimi digiti. The palmaris brevis contracted well, and the lumbricals, even the 3rd and 4th, fairly. The interossei in the 4th space act but very slightly indeed. The ring finger is faintly abducted and the little finger adducted. The other small muscles of the hand act fairly.

The voltaic current from 12 cells produces weak and *tardy* contraction of the abductor minimi digiti, but ACC>KCC, while for all the other muscles of the hand supplied by the ulna KCC equalled or was greater than ACC. This reaction of degeneration was very distinct. Neither KOC nor AOC could be obtained with a current from 22 cells, which was as strong as the patient could bear. With a large plate electrode applied to the back of the forearm and a small one to the motor point for the abductor minimi digiti, a strong kathodic current from 20 cells, becoming diffused, caused contraction of all the small muscles of the hand; but the anodic current produced hardly any perceptible movement except in the abductor minimi digiti, which contracted strongly.

The skin of the affected area was quite insensible to galvanic or faradic currents of moderate strength. The muscles on the ulnar side of the forearm appeared wasted, but the flexor carpi ulnaris and flexor profundus digitorum contract with the faradic current, and with the voltaic KCC > ACC.

June 8th.—The abductor minimi digiti now contracts to a strong faradic current. Sensation is returning, and he can perceive a touch half-way up the ulnar side of the little finger. Voluntary motion is also improving, and he can slightly abduct the little finger.

All the conditions continued to improve, but the patient went into another county, and was lost sight of before the restoration of motion and sensation was complete.

Case II.—The next case I need not enter into at such length. It relates to a policeman, G. C., aged 23, a very powerful man, weight 18½ stone, and 6 feet 3¾ inches in height. He was admitted on Nov. 24th, in the third week of a moderately severe attack of enteric fever. He had at first sharp diarrhœa, but no throat affection. There was no complication, and he convalesced rapidly. The highest temperature since his admission was 103. There was no albuminuria.

On Nov. 28th he complained of *stiffness* of the ring and little fingers of the left hand. On Nov. 30th there was visible wasting of the muscles of the thenar and hypothenar eminences, and they were soft and flabby to the touch. There was slight twitching of the flexor brevis pollicis and of the abductor pollicis. There was loss of common sensation in the little finger, half the ring finger and the ulnar side of the hand, nearly as far as the head of the ulna; but no pain, tingling, or numbness.

With the hand at rest in the supine position, the ring and little fingers are semiflexed and cannot be straightened voluntarily; neither can they be completely abducted. There was no interference with flexion, or passive motion; no tenderness along the course of the ulnar nerve; and no affection of the other hand. The muscles of the left thenar and hypothenar eminences responded to the faradic current, but feebly when compared with the right hand.

Dec. 28th.—Sensation unaltered, wasting increased, and affecting also the adductor and flexor brevis pollicis. Power of movement less; little finger cannot now be either abducted or adducted. No appreciable alteration in temperature.

Jan. 4th, 1888.—He became an out-patient to-day, and a complete electrical examination was now undertaken.

Faradic current.—Electro-sensibility much diminished in the affected area: muscles of the ball of the thumb contract fairly; the opponens well. Muscles of the little finger do not contract at all to a very strong current, sledge at 7 cm., though in the right hand they contract strongly with sledge at 8 cm. distance.

Voltaic.—Electro-sensibility diminished in the affected area, *but is greater for positive closure (A.C.) than negative (K.C.)*. Muscles of the little finger contract very poorly to a current of 40 milliampères and ACC > KCC. He cannot yet voluntarily abduct the little finger.

Jan. 28th.—Patient resumed his duty as a policeman.

Feb. 11th.—There is still a very marked difference between the size of the muscles in the two hands: this is partly due to the increased volume and tone of the muscles of the right hand, which are now large and hard. The tone of the muscles of the left hand has improved, though the volume has not increased much. Sensation is still somewhat impaired in the little and ring fingers, but has returned more to the ulnar side of the hand. Patient can now abduct the little finger fairly, but not so well as the right, neither can he yet completely extend it. Sometimes in putting on a woollen glove the little finger remains flexed without his knowledge, till it is discovered by sight.

He still further improved, and eventually got quite well.

CASE III.—The last case refers to L. W., a lad of 15, an errand boy, who was admitted on Nov. 2nd, 1887, suffering from enteric fever, already of about ten days' duration. Two sisters were in the hospital at the same time with enteric fever, another sister in the Children's Hospital, and the father and a fourth sister were lying ill with it at home. Thus the case was one that originated in a very active and widely spreading poison. The primary attack lasted 60 days, during which the temperature only twice fell to 100°, and for a long time remained between 103° and 105°. The highest point reached was 105° 8. There were numerous crops of spots, marked albuminuria, and slight diarrhœa. For the first fortnight he had a sharp attack of laryngitis, indicated by hoarseness, aphonia, and cough, with pain in swallowing. The fauces were deeply congested, but no ulceration or exudation could be seen. He was too ill for the laryngoscope to be easily used.

After a commencing convalescence he had a relapse lasting eight days, the highest temperature being 104°·6; and again after nine days' convalescence another relapse of thirteen days' duration, with a temperature of 105°. Fresh spots came out on each occasion. Of the treatment I need say nothing except that he had several cold baths during the primary attack.

On Jan. 4th, in the midst of the second relapse, he said that he began to feel pain in the little and ring fingers of the right hand, and along the ulnar border of the hand, but he did not mention it till Jan. 14th. On the latter date there was loss of sensation in the parts supplied by the ulnar nerve, with marked *tenderness* on grasping the muscles of the little finger, or of the ball of the thumb, and on pressing the arm along the course of the ulnar nerve, nearly as far as the axilla. The little and ring fingers cannot be abducted at all, neither can they be fully extended, but remain always partially flexed. The thumb can be moved in all directions, but imperfectly. There is evident wasting of the muscular mass along the ulnar border of the hand, and in the first interosseous space. The left hand is unaffected.

For some weeks past there has been *tenderness of the toes* of both feet, so that the toe nails could not be cut on account of the pain it caused in the nail bed and in the pulp at the end of the toes. In three other cases recently I have met with this condition of pain in the toes, and in one of them in the arms also. In one instance the tenderness of the feet was so great, that they had to be protected from the pressure of the bed-clothes by a cradle. But in none of them was it followed by muscular wasting or definite loss of sensation, so far as I could detect.

Feb. 3rd.—The tenderness of the toes is quite gone. Sensation has returned somewhat to the affected region of the right hand, but is still much impaired along the ulnar border. The tenderness on pressure has disappeared. The power of movement is improving, and he can now hold his knife at meals, which he has hitherto been unable to do. He has had considerable dilatation of the heart, and a very irregular pulse. Another sign of lowered tissue nutrition is the formation of numerous cicatrices (lineæ albicantes) in the skin above each knee (Fig. 2), of the same

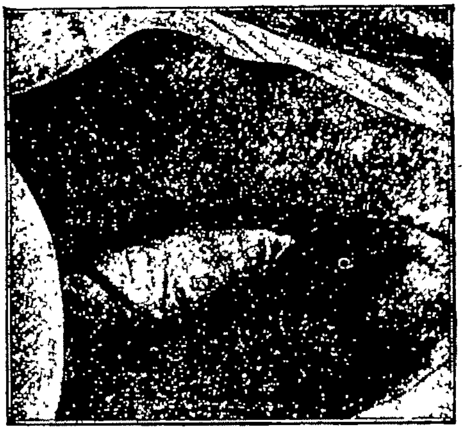

FIG. 2.—Taken directly from a photograph from an "untouched" negative. Lineæ albicantes above and below the left knee and above the right

nature as those observed on the thigh in stout people in the region of the tensor vaginæ femoris, and on the abdomen during pregnancy. The patient used to lie "curled up," with his knees nearly up to his chin, and the affected portion of skin was stretched. I have observed similar marks above and below the knee in another case, a girl of 17, who used to lie "curled up." It was an extremely severe case.

Feb. 17*th.*—Photograph taken (Fig. 3). There is evident wasting of the interossei, shown by the hollowness between the metacarpal bones on the back of the hand. Electrical examination carried out to-day. *Faradic.*—*Left* hand, all the muscles contract well with sledge at 9 c. distance; *right*, opponens and abductor pollicis contract with sledge at 5 c., but none of the other muscles of the hand. This was as strong a current as could be borne. *Voltaic.*—With a weak current (8 cells), the adductor pollicis and abductor minimi digiti contracted very slightly, and KCC=ACC for the thumb muscle, but KCC<ACC for the little finger.

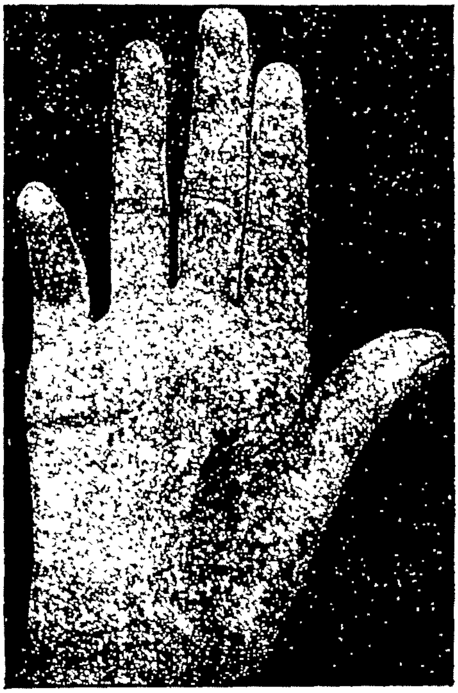

FIG 3.—Taken directly from a photograph from an untouched negative. The ulnar border of the hand is concave from muscular wasting. The little finger abducted, and in "claw-hammer" position. The ring finger is in a similar condition, but less marked.

March 3*rd.*—The power of movement has not improved much, though sensation has largely returned. The muscular wasting is more evident now from the recovery of the normal state of nutrition of the unaffected muscles of the same and of the opposite hand. The little and ring fingers remain in a marked "claw hammer" position (Fig. 8), from the paralysis of the interossei and two inner lumbricals. The fact of the two outer lumbricals, supplied by the median, being unaffected, is sufficient to prevent this position in the other fingers, although the interossei are affected.

April 20th.—Muscular wasting still very evident.

If these cases, which were so plainly examples of neuritis of the peripheral part of the ulnar nerve, were due to the specific poison of enteric fever, it is, to say the least, surprising that the distribution should have been so exactly the same in each case, and that there should have been no sign of neuritis affecting any other region. It is true that in the last case there was the pain in the toes, mentioned as having been met with in several other patients also, in whom the ulnar nerve was not affected. It is quite possible this may have been a slight and transient neuritis. Two of the three cases were exceedingly severe, and were each complicated by a throat affection, which, however, was not at all of a diphtheritic nature. In the first and last cases the patients were accustomed to lie "curled up," with the elbows sharply flexed for days together, and each chiefly on the side eventually affected. It is quite possible that thus a neuritis from over-extension was set up, and that the earlier numbness and tingling were disregarded during the delirious condition. And further I can hardly conceive a more favourable condition of tissue malnutrition than these two cases presented. None of these conditions, however, apply to the policeman. His attack of fever was not much above the average in severity; he had no throat affection, no great loss of flesh, and lay always on his back, with the arms generally extended.

On the whole, I think there is evidence that traumatism plays a part in the etiology of these cases, though probably tissue malnutrition may be an essential condition.

Critical Digest.

RECENT RESEARCHES ON GENERAL PARALYSIS OF THE INSANE.

BY T. DUNCAN GREENLEES, M.B.

THOSE who have studied the ætiology of general paralysis are unanimous in the opinion, that it is a disease essentially the result of modern civilisation, and among its more frequent causes are to be found mental worry or anxiety, the effects of the high pressure at which we live in the present age—the "surmenage" of French authors—this factor being associated, in many cases, with dissipated habits. When we come, however, to discuss more fully the details of its ætiology, we find that differences exist among observers as to the importance and frequency of the various causes, and this diversity of opinions is to be explained by a reference to the sources from which the information has been obtained. Thus in rural districts the same causes do not hold good to the same extent as they do in populous districts or large cities; the anxious struggle for existence and the desire to attain to wealth rapidly is rarely met with in agricultural counties, where, apart from moral influences, the most important and frequent cause of general paralysis is intemperate habits acting on a brain peculiarly sensitive to the effects of alcohol. The form of insanity affecting any individual is always more or less influenced and modified by the temperament of that individual during mental health, and this fact may assist to explain the frequency of the maniacal and exalted form of general paralysis in those subject to the worries of city life, as contrasted with the melancholic or demented type so common in the agricultural or labouring classes.

In an interesting statistical enquiry[1] on the ætiology of general paralysis, Dr. Christian gives the result of his observations in 340 cases—all males, and derived chiefly from an urban population. The age at which general paralysis is said most frequently to occur is between 30 and 50; in 172 cases the author found the disease to commence between 40 and 50. This confirms Bayle's view, "that general paralysis occurs at that time of life when the individual enjoys the fulness of his faculties and functions, when he is especially ruled by ambition, and consequently exposed to all kinds of disappointments and troubles."

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[1] "Recherches sur l'étiologie de la Paralysie Générale chez l'homme," par Dr. Jules Christian: 'Archives de Neurologie,' Sept. 1887, p. 205.

Christian found that married men are most liable to the disease, and that half his cases were persons of "very low intellectual culture." In another place he states that, among soldiers, officers are most liable to the disease—not very complimentary to the "intellectual culture" of French military officers. During the siege of Paris, in 1870–71, the number of soldiers admitted to Charenton suffering from general paralysis was very considerable. Contrary to the observations of other writers, he maintains that heredity has an important influence on the ætiology, for, apart from cases regarding which he was unable to obtain reliable information, 35 per cent. had a distant hereditary neurosis.

While carefully differentiating the symptomatic relations between insanity, the result of chronic alcoholism, and general paralysis, the author finds that the latter disease is rarely the result of alcoholic intemperance when this cause is taken alone, and he states that, as a result of his own observations, alcoholic and venereal excesses, when considered as causes of general paralysis, have been much exaggerated in the past. Syphilis may produce symptoms of the same nature as those common to general paralysis, although syphilitic cerebral disease must not be confounded with general paralysis: in 23 of his cases there was a previous history of syphilitic infection, but the symptoms in these were ameliorated under appropriate treatment. Ten cases were due to sunstroke and six were the result of cranial injuries. With regard to previous bodily ailments, he lays some stress on the importance of this factor as bearing on the ætiology. This is a cause which, in the author's opinion, has been much neglected by previous writers, and he contends that an antecedent illness, by giving rise to a general feebleness of the body, renders the brain, as well as other organs, more susceptible to influences which, under other circumstances, would have no deleterious effect.

There are many points in the pathology of general paralysis regarding which we are still in doubt: the older authorities, such as Bayle, thought it was a distinct disease of the nervous system, more especially of the brain: but, on the other hand, Pinel, Esquirol, &c., maintained that it was merely one of the complications or terminations of insanity. Since the publication of cases presenting all the motor symptoms without any mental changes, writers are more inclined to view it as a disease of the cerebrospinal nervous system, always presenting motor symptoms and generally accompanied by psychic manifestations. Although the pathology of general paralysis is but imperfectly understood, the morbid anatomy has been thoroughly worked out both by British and foreign investigators. The morbid anatomy of general paralysis may be briefly summed up as follows:—A thickened, inflammatory condition of the pia mater with more or less adhesion to the subjacent gyri—generally in scattered patches and affecting one hemisphere more than the other; a corresponding destruction of the grey cortex to a variable extent; a general atrophy of the convolutions, more especially over those areas where the inflamed condition of the pia is most marked: there are changes in the cerebral blood-vessels, the walls of the minute arteries are thickened, the vessels are frequently tortuous and their outlines irregular. The perivascular spaces are dilated and contain *débris*. The large pyramidal nerve-cells of the cortex are generally atrophied, their processes shrivelled and the contour of the cells rounded, their contents being replaced to a variable extent by altered fatty or pigmentary particles. Finally, the neuroglia is increased in amount, compressing both vessels and cells, and the neurogliar nuclei are proliferated. All these changes affect not only the brain, but likewise the spinal cord.

With these data to hand, it can easily be understood that there are many theories in existence with regard to the pathology of this interesting disease. Presenting indications of both an inflammatory and a degenerative origin, each of these conditions has its advocates. Among the more recent contributions to this subject may be mentioned an article by Dr. Leah.[1] in which he maintains that general paralysis originates by prolonged irritation in the cortical cells, and spreads in the direction of the nerve paths to the corresponding cortical region of the opposite side. He then attempts to prove that it is essentially a parenchymatous disease (in opposition to those who maintain that it has an inflammatory origin affecting primarily the neuroglia) and differs in no respect from the changes observed in such a condition as that found in anterior polio-myelitis.

He concludes an interesting article as follows—

(1.) The usual order in which the cerebral functions fail is first the mental, next the motor, and still later the sensory.

(2.) This order of functional failure corresponds with the mode of distribution of the morbid changes, and with the probable physiological relations between these parts and the organ of mind.

(3.) Cases of early and marked failure of the cortico-sensory region are most likely to occur among those in whom these centres have been most under control, and in whom therefore the nervous paths from the mental organ to the sensory have been most cultivated.

(4.) When the motor affection follows the mental it comes on at an early period; but when the motor affection is the primary, mental symptoms only follow after a long and uncertain interval.

(5.) If the above statements be true, they are strongly suggestive of propagation of irritation and disease along nervous paths in the direction of nervous impulses.

(6.) There is reason to believe that the disease usually begins in a unilateral focus, and probably, generally in the right side, and corresponding parts in the left hemisphere are involved by a process akin to sympathetic inflammation.

The condition of the spinal cord in many cases of general paralysis is such as to induce many observers to believe that the disease is essentially spinal in its origin, or at least that the morbid state of the cord plays an important part in the pathology of the disease. Of this there can be no doubt, and recent investigations have thrown considerable light on the close relationship existing between the cerebral and the spinal changes in this disease. Dr. Stewart, of Glamorgan Asylum, in a series of observations on the spinal cord of the insane,[1] examined the cords of five general paralytics. In four of these five cases there was bilateral disease of the lateral columns. In only one case were the external divisions of the posterior columns so affected as to give rise to ataxic symptoms during life. The columns of Goll were diseased in four cases, and in one the spinal cord as a whole was atrophied, its weight being only 15½ drams. The author quotes Quain, who gives the weight of the spinal cord in a healthy adult at from 16 to 28 drams; and Boyd, who found the average weight of the cord, in those dying insane, to be 19½ drams. In one case Dr. Stewart found a general condition of spinal sclerosis as well as other microscopical changes. These changes, which were found in all his cases to a variable extent, consisted of an atrophied condition of the multipolar cells of the grey matter with a fatty pigmental degeneration of the cell-contents to a variable amount, together with a tortuous engorged condition of the minute blood-vessels, some of which presented localised dilatations along their course. At the same time he found alterations in both the dura spinalis and the pia mater of the cord, similar in nature to those usually affecting the same membranes of the brain.

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[1] "A New View of General Paralysis of the Insane." 'Birmingham Medical Journal,' 1886.

The peculiar condition of the cerebral membranes, the so-called "Pachymeningitis interna hæmorrhagica" of German authors, has been carefully studied by Dr. Wiglesworth,[2] who states his opinion that the lesion is not the result of inflammation at all, as had been previously thought, but is solely due to effusion of blood under the dura mater: the hæmatomata thus formed becoming organised and eventually converted into fibrinous membranes; and, on account of the convolutional atrophy, so frequent in general paralysis, resulting in loss of the necessary support to the meningeal blood-vessels, this pathological condition is most frequently met with in cases of this disease.

General paralysis is one of those diseases that seem to exercise a marked influence on all the organs of the body, and the condition of the viscera in this disease has received some attention from scientific alienists. Whether this morbid influence is the result of the long-continued excitement, or whether it is the result of distinct neurotic changes in the organs themselves, it is difficult to say. The writer has shown[3] that in 33 cases of general paralysis, the heart was found distinctly hypertrophied in 39·3 per cent., and valvular disease existed in 13 cases. An explanation of this morbid condition of the heart is to be found in the atrophy of the brain and thickened arterial walls offering a certain amount of opposition to the flow of blood; and, in fact, the changes in the circulation of a general paralytic are identical in many respects with those that occur in old age and chronic Bright's disease. This theory is further verified by the characters of the pulse-tracing at different stages of the disease.[1]

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[1] "Observations on the Spinal Cord of the Insane:" 'Glasgow Medical Journal,' 1886.

[2] "On Hemorrhages and False Membranes within the Cerebral Subdural Space:" 'Journal of Mental Science,' Jan. 1888, p. 509.

[3] "Diseases of the Circulatory System in the Insane:" 'Journal of Mental Science,' Oct. 1885.

Dr. D'Abundo[2] has recently contributed an important article on the condition of the Urinary Bladder and Prostate in General Paralysis. In most of his cases, even although there was no marked disease of the urethra or prostate, he found evidences of cystitis, and examination revealed hypertrophied condition of the vesical walls with general dilatation of the viscus—no doubt the result of muscular atony. He explains the prostatic hypertrophy, observed in several of his cases, to be due to increased functional activity of the gland, and suggests a close connection, as to cause and effect, between the chronic cystitis and the enlarged prostate, thus throwing some doubt on the generally accepted theory, that the former is consecutive to the latter.

M. Baillarger[3] has published an interesting article on the analogy of the symptoms of Pellagrous insanity and general paralysis of the insane, in which he shows that, in many cases, it is well-nigh impossible to arrive at a satisfactory diagnosis. He quotes M. Roussel, who states that in the Pellagrous patient there is a muscular debility which, when it becomes aggravated, constitutes Pellagrous paralysis accompanied by dementia, the progress of which follows that of the paralysis, and that this condition is associated with the characteristic cachexia of the disease. Although very different in the early stages, Pellagrous mania and paralytic mania have this in common, that they end in paralytic dementia, and, as the dementia succeeds to the mania, the muscular weakness becomes predominant.

The paresis in the Pellagrous patient appears first, and is always more pronounced in the inferior extremities: Lussana and Frua explain this by stating, that the weakness attacks the muscles of the legs first, because they support the weight of the body: many years ago Calmeil had observed the same fact in general paralysis, and had given to it the same explanation.

The general paresis of the Pellagrous patient begins as a muscular weakness, its course is slowly progressive, but it always remains incomplete: the writer points out that these characteristics are exactly those of general paralysis of the insane, and he proceeds to consider the differential diagnosis between the two diseases, stating that the connection between them may be aptly compared to the relationship existing between general paralysis of the insane and chronic alcoholism. Roussel is again quoted, who, after explaining the symptoms common to Pellagrous insanity and general paralysis, proceeds as follows:—"It is therefore in the particular characteristics of the impediments of speech and in those of the delirium, and finally in the situation that one must seek the more important elements of the differential diagnosis. The speech of the Pellagrous patient is slow, difficult, and hesitating, but the author has never observed that accentuated trembling—that kind of singing—that effort to 'cut the words,' which has been described by alienists. Of course it is understood that in the beginning of general paralysis, when the difficulty of speech is but little pronounced, it is only with much attention that one can recognise surely the differential shades."

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[1] "Observations with the Sphygmograph on Asylum Patients:" 'Journal of Mental Science,' Jan. 1887.

[2] "American Journal of Insanity," 1887, p. 340 (*translation*).

[3] "Analogie des Symptômes de la Paralysie Pellagreuse et de la Paralysie Générale Diagnostic différentiel," par M. le Dr. Baillarger.

Taking the above quotation as his text, M. Baillarger discusses the differential diagnosis under three heads:

(1.) The particular characteristics presented by the impediment in the speech;

(2.) The nature of the delirium; and—

(3.) The situation of the paralysis.

With regard to the speech-derangements, he draws particular attention to the fact, that the difficulties of speech, the result of ataxy or of paralysis of the speech-muscles, must not be confounded with that hesitancy due to the intellectual state of the patient, for in Pellagra the patients are generally melancholic (with stupor) or demented, and they answer questions slowly and with some hesitation. At the same time, in the minority of cases, speaking is accompanied by a tremulousness of the lips, so that it is utterly impossible to diagnose such a case from the early stage of general paralysis. As the result of observations of several cases of Pellagrous insanity under his care, and which he details fully, he concludes his article as follows—

"(1.) The symptoms of Pellagrous paralysis offer the greatest analogy with those of general paralysis. Indeed in both diseases the paralysis is progressive and general, and remains incomplete till the end.

"(2.) One can observe in Pellagrous mania, as also in paralytic mania, the hesitation of speech with convulsive trembling of the lips preceding articulation of the words, and besides, sometimes inequality of the pupils. As these symptoms can exist in both diseases, they do not therefore form an important point in the element of diagnosis.

"(3.) When one notices impairment of the speech-function in Pellagrous mania it is principally by the nature of the excitement that one can distinguish it from paralytic mania.

"(4.) One can observe constantly in paralytic dementia of the insane an embarrassment of the speech clearly characterised; that is to say, according to writers this impediment of speech does not exist in Pellagrous paralytic dementia; consequently it seems to me that this symptom must furnish the principal element in the differential diagnosis between the two diseases."

Dr. Tuczek, of Marburg, has recently contributed an important paper on Pellagrous insanity, as the result of his observations of 350 cases and 8 autopsies. The pathological appearances, both of the brain and spinal cord, seem to bear a close analogy to those frequently found in general paralysis, and the peculiar nervous disturbances, as found in Pellagra, he believes to be due to the prolonged use of diseased maize, his investigations pointing to the disease having a toxic origin.

As in tabes dorsalis and other nerve degenerations, so in general paralysis of the insane we occasionally find skin eruptions and ulcers, in all probability due to trophic nerve changes. M. de Montyel has recently published an article,[1] in which he maintains that anæsthetic ulceration of the plantar aspect of the foot is much more common in general paralytics than has been previously recognised. He summarises his observations as follows:—

(1.) Progressive general paralysis is a cause of perforating ulcerations as much as other diseases of the nervous system.

(2.) The scarcity of observations published on perforating ulcerations in cases of general paralysis is perhaps explained by the fact, that this complication can only be discovered by very careful examination, and that, in the past, attention has not been sufficiently directed to the condition of the feet.

(3.) Perforating ulcers are to be found chiefly in those general paralytics whose illness was due to alcoholism, or who have been known to have indulged freely in drink.

(4.) The perforating ulcer, to judge by the facts known up till the present time, seems to favour the remissions in general paralytics who are affected by it.

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[1] "Du Mal perforant dans la paralysie générale progressive," par le Dr. de Montyel: 'L'Encéphale,' 1888, p. 257.

Reviews and Notices of Books.

Ueber der Shock. Eine kritische Studie auf physiologischer Grundlage. Von Dr. G. H. GROENINGEN. Wiesbaden, Verlag von J. F. Bergmann. 1885.

THE very excellence of this book has, to some extent, prevented an earlier notice of it in the pages of this Journal. It is a book which ought to be translated into English, so as to find a place in the library of every surgeon to whom German is an unknown tongue, being by far the ablest and most exhaustive treatise which has yet appeared—whether separately or as part of a more general work—upon the subject of "Shock," full alike of interest and instruction. The opening pages are devoted to an historical *résumé* of all that has been written on Shock, from the time when the word Shock, at the middle of the last century began to be used as a convenient phrase for indicating the symptoms which result from any sudden and severe injury, to the time now, when it has been incorporated into the German language as the most appropriate expression for that combination of phenomena and symptoms with which all are familiar.

The absence of any precise pathology of shock has always surrounded the subject with much difficulty, nor is this lessened by the number and variety of the causes of shock, nor by the fact that the symptoms by which the condition is recognised, and which indeed make up the condition, spontaneously disappear, and that no morbid lesion satisfactorily accounting for the symptoms has been found in the thousands of cases which have ended fatally. The field has thus been open for speculation and hypothesis, and quoting from Blum, the author says, "Le raisonnement seul, avec l'aide de la physiologie expérimentale, permettra de résoudre le problème de la nature du shock." Opinions have ever leant to the view, that the symptoms of shock are most probably due to some functional disturbance of certain systems, and the charge has been laid in turn to the nervous system and to the circulatory system. The chief hypotheses are subjected to a critical examination by the author, who sees grave objection to regarding the phenomena of shock as dependent on reflex paralysis of the heart alone, a theory which takes but small account of the muscular prostration, the diminution of common sensibility, and of reflex action, the general apathy, the relaxation of the sphincters and other like phenomena. He rejects moreover, the theory that shock is due to reflex paralysis of the vessels generally, whereby the circulation is enfeebled. True it is that feebleness of circulation is one of the pronounced signs of shock, but alone it is inadequate to explain the whole, and observation shows that the state of the circulation varies very much in different cases. He objects also to those hypotheses which attribute shock to disorder in some only of the functions of the nervous system, especially that which calls in the aid of inhibition, and an effect on the heart of a supposed irritation of the vagus. "Niemand weiss," the Author writes, "was eigentlich hemmt, und wie es hemmt."

Discarding all previous theories as inadequate—though each of them has in it the merit of accounting for some of the symptoms, the Author says that a step higher must be taken, if we would find an explanation of all the phenomena, and shock, he proceeds to tell us, is a depression or exhaustion of the spinal cord and medulla oblongata brought about by any sudden impression or injury. The nervous system shows the insult inflicted on it by a suspension of its functions. It suffers somewhat in the same way as the delicate mimosa, which hangs its head when the plant has been too roughly handled. It matters not what is the precise term employed to designate this state of things, suffice it that there is extreme exhaustion—whether due to a molecular or to a chemical change no one can say—induced by the violence of the irritation or insult. It is very much a question of degree. The effect upon a sensory nerve varies according to the amount of the irritation, varies from a mere sense-impression of the irritation upward to that of pain, and still further may the irritation be carried, both in amplitude and degree, until there follows the state of shock, which is the last and most extreme result, now become general, and affecting central parts of the nervous system. And this last effect is exhaustion of the spinal cord and medulla oblongata, and thereafter ensue the symptoms which are recognised as those of shock.

The question occurs to us, however, why the brain itself should be exempted from this state of exhaustion which the author attributes to the cord and medulla. Not the least obvious amongst the symptoms of shock are those which clearly depend on brain enfeeblement, and although this may be largely due to the fact, that in the state of shock the brain is being but poorly supplied with blood, yet nevertheless the occurrence of shock from purely psychical causes shows that the cord and medulla may be affected through psychical channels, and that there is in all probability exhaustion of other parts of the nervous system than those which are alone included in the Author's definition. The Chapter on Psychical Shock is extremely interesting, but we think that hardly sufficient importance is attached to this. the psychical element, in the induction and maintenance of the symptoms of collapse. This psychical element explains, to some extent, the differences in the degrees of shock which are seen after injuries apparently the same. It were strange if the nervous systems of all were cast in precisely the same mould. We know indeed that they are not, and it is to individual differences that many of the different effects of the causes which conduce to shock are in the end to be traced. All sorts of functional disorders of the nervous system—spasms, pareses, anæsthesiæ, are brought about by sudden fright, for example, and the functional derangement which is of a wider range, and which by its symptoms is recognised as shock, may be induced by a similar cause, but induced in very varying degrees of intensity. Physical and psychical causes probably are both at work in most cases of shock, and every part of the nervous system shares in the exhaustion. Under our observation some little time ago, was a woman admitted into the Hospital in a grave state of collapse after a severe burn. She rallied well, under the influence of perfect quietude, warmth, alcohol and digitalis, and gave good promise of recovery, when on the fifth day her husband bluntly announced to her that he and his children were all going into the workhouse. The woman instantly became collapsed, but this time there was no rally, and in a few hours she was dead. The influences which were here at work may exert themselves in a smaller degree and with less serious result, but we do not doubt that they play a considerable part in determining the onset, duration and character of the symptoms of shock, whatever may have been the original physical injury.

Mere bodily injury alone may at any rate be insufficient to cause shock, and we have in mind in saying so the case of a man whose thigh was amputated within two hours of his accident, his leg having been crushed to pieces by a falling wall, and in whom there were at no time any of the symptoms which are known as those of shock. His absolute indifference to the injury and its consequences was at the same time most remarkable. Exhaustion of the spinal cord and medulla affords, it seems to us, an insufficient explanation of all the phenomena and of the many differences in their presentation, and we should probably be more correct in attributing the symptoms of shock to exhaustion for a time of each and every part of the nervous system, remembering always that there is a personal element—there is the particular nervous organisation of the individual—to be considered in every case. No one really knows what shock is, and we have to judge of it by its symptoms, and become familiar with the clinical picture which it presents. In an age of pathological inquiry, we are prone to be dissatisfied when no certain pathological lesion or changes can be established. It is, however, very doubtful if there is, or if there will ever be found, any structural pathological change to account for the symptoms. The change is in dynamics and in function, and the underlying structural changes, if there be any, whether they be molecular or chemical, can never be known, being discoverable only when investigation of the requisite kind is impossible, and that is during life. The cessation of life at once transforms the whole machinery, and for ever closes the door to fruitful inquiry. Not so with experimental physiology, which may in time elucidate the whole matter.

Our business here, however, is not to say what we may think on the subject of shock, but rather and much better to speak of the merits of this excellent book by Dr. Groeningen. From every conceivable point of view, and from every side, he seems to have dealt with the subject; with shock as it affects the old and young, shock in its relation with operations, with the narcosis of anæsthetics, after the different injuries to which various parts of the body are liable, with shock from fright, with that which is seen after railway collisions, with it in its medico-legal aspects. The symptoms due to brain-concussion he does not regard as those of shock, and he looks on pain as in no sense a cause of it.

He excludes from the category of true shock all those cases in which collapse with fatal ending supervenes some days after the infliction of an injury, and where there has been an intervening period of progressive improvement and well-doing. Septicæmia is by far the commonest cause of such conditions, and is happily seen not nearly as often as it was. And here also is much more properly allied the so-called "prostration with excitement," about which much has been written, but which has probably no true relation to shock. This again is not nearly as common as it was, if we may judge from the writings of a previous age. He contends that the so-called "reaction" after shock is by no means invariably present. We are disposed to agree with him, though rather inclining to the view, that the period of reaction may be very short indeed; the individual nervous organisation having a considerable influence in determining the slow or speedy restoration of the nervous equilibrium.

Not the least interesting of the chapters of the book is that on Treatment. The Author thinks highly of subcutaneous injection of strychnin, and speaks with approval of belladonna and digitalis. The last of these drugs we have ourselves thought to be of distinct use in some cases; but the surgeon will act wisely in having "the courage to do little," and rely on perfect quietude, both of mind and body, the maintenance of the animal warmth, together with the most careful administration of food and stimulants. With what exceeding care, and for what length of time the treatment must be carried out, could not be better shown than in the case of the burnt woman to which we have referred. The exhaustion and prostration of the nervous system, whether of the whole or of parts of it, pass away only by degrees, and it is only by lapse of time that the nervous tone which controls, presides over and directs the various bodily and mental functions, can be re-established.

The work ends with a series of "conclusions," which, to the number of 108, give in few words the pith of the whole matter, and are not the least valuable part of the book. There is, moreover, a bibliography, which for exhaustive completeness can hardly find an equal in any treatise of the kind. Enough has been said, we think, to show how high is our estimate of this work. Clear, concise, and judicial in tone, it ought to have been brought much earlier to the notice of our readers.

HERBERT W. PAGE.

Die Entzündung der peripheren Nerven (Polyneuritis, Neuritis multiplex).—Two Lectures by Prof. E. Leyden. Berlin, 1888.

The first of the two lectures deals with the general subject of inflammation of the peripheral nerves, and is largely taken up by an historical sketch. Most of it is already pretty generally known. He relates at length the two cases published by him in 1879–80. About these I would only notice the following points. In the first case there was swelling of the limbs, and considerable œdema, indicating marked general tissue change. At the autopsy the alteration of the nerves was chiefly limited to the region of the elbow-joint. In the second case, which died in an earlier stage, no mention is made of swelling or œdema, but the changes in the nerves were again confined to the regions of the elbow and knee, and especially affected the peroneal nerve. This is not quite in accordance with the general statement, that the degeneration is most marked at the periphery and diminishes towards the centre, and that the main trunks are seldom affected. The fact that in these cases, which were so thoroughly examined, the neuritis was limited to the regions of the joints, where the nerves are so exposed to injury, either direct or from cold or by tension, deserves more attention than it appears to have received, and suggests that traumatism in some form may play a part in the etiology.

Among the less frequent symptoms of multiple neuritis the author mentions some which are not without significance, because of the difficulties they raise in the diagnosis from paralysis due to cord or brain disease—a diagnosis of some practical importance, on account of the very different prognosis in the two classes of cases. These uncommon conditions are, implication of the facial nerve, strabismus, nystagmus, dilatation or fixity of the pupil, paralysis of the sphincter of the bladder or of the rectum, delirium, irritability, and sleeplessness. The author considers cardiac complications of much importance. These consist of palpitation, increased frequency of the pulse, anxiety, and signs of weakness of the heart. They are especially frequent in diphtheritic paralysis, and in the alcoholic form, but are also met with in spontaneous cases and especially in Kak-ke (Beri-beri).

As regards treatment Professor Leyden says that it should be chiefly hygienic and dietetic. Etiological treatment is usually of benefit only in special forms, such as rheumatic neuritis, and that due to lead and to alcohol. For the first (and perhaps for several other forms) salicylate of soda, antipyrin, or antifebrin generally relieve pain, and sometimes bring about great improvement. For lead paralysis the iodides and sulphur are beneficial. For the alcoholic form abstinence from the specific poison is requisite. In symptomatic treatment the chief necessity is to relieve pain, and in this respect Professor Leyden warns against the use of morphia, which in this class of diseases is especially liable to be abused.

Bathing and rubbing, particularly in the earlier stages, he thinks usually do more harm than good by disturbing the needful rest, and increase the pain by the necessary movements. Electricity he considers essential for exact diagnosis and prognosis, but not of much value in treatment. Rest is the all-important factor.

In the second lecture Professor Leyden points out that, through the recent additions to our knowledge, multiple neuritis must be considered as a *Group of Diseases* and no longer as a single disease. He gives the different varieties in the group in a tabular form.

1. The *Infectious Form*, comprising neuritis after diphtheria, typhoid, and other infectious diseases: primary infectious neuritis, Beri-beri: neuritis in syphilis and in tuberculosis.

2. The *Toxic Form*, comprising paralysis from lead, arsenic, phosphorus, carbonic oxide, ergot, mercury, and alcohol.

3. The *Spontaneous Form*, coming on after over-exertion and after unusual exposure to cold.

4. The *Atrophic* (*dyscrasic*, *cachectic*) *Form*, in connection with anæmia (pernicious anæmia), chlorosis, cachexia, marasmus, the cancer cachexia. Diabetes (tuberculosis, Beri-beri).

5. *Sensory Neuritis.*—Pseudo-tabes.
   (*a.*) Multiple neuritis affecting sensory nerves.
   (*b.*) The neuritis of the sensory nerves in tabes dorsalis.

Seeing that multiple neuritis is associated with such various diseases, a very important question arises: viz., whether in the different forms of neuritis we have to do with the same morbid process. Professor Leyden thinks not. All the forms resemble one another in this, that the cord, nerve-roots, and main nervous trunks are unaffected, and the neuritis is confined to the smaller nerves, distant from the cord and not depending on any alteration in the latter. But the form of affection of the peripheral nerves appears to be in some cases distinctly inflammatory, in others dependent on preceding hæmorrhage, and yet in others a passive degenerative process.

Before discussing the various forms of multiple neuritis Professor Leyden calls attention to the probability of acute ascending paralysis, or Landry's paralysis, proving to be due to neuritis. He has only had the opportunity of examining one case *post mortem*, and in that no change in the cord, nerves, or muscles could be demonstrated.

The infectious form of multiple neuritis usually affects the motor nerves, but occasionally the sensory, and then the case is described as acute ataxia or pseudo-tabes.

With regard to the intimate cause of the neuritis in infectious diseases, Professor Leyden raises the following very important question: Are we to suppose that the pathological microbes become localised in the affected nerves and set up the neuritis? This is unlikely on several grounds. Specific micro-organisms have not yet been found in the affected nerves. Also the period at which the neuritis occurs, and the course of it, render it most unlikely that it is due to the specific micro-organisms of the infectious disease. Professor Leyden thinks it much more probable that some irritating substance, possibly a ptomaine, produced during the course of the disease, is the true cause. In this he agrees with Rosenheim,[1] who says, "Not an organised virus, but some deleterious chemical substance produced by bacteria, is the cause of the infectious form of multiple neuritis." On this point they agree very much with Fürbringer, who attributes the nephritis of infectious diseases not to organisms, but rather to some chemical substance, probably in the nature of a ptomaine.

This view of the origin of the infectious form of neuritis gains great support from the fact, that a toxic neuritis is known to occur. It simplifies greatly our conception of the morbid process if we suppose, that in multiple neuritis there is generally (if not always) a local irritant: in the infectious form, a ptomaine; in other cases, alcohol, arsenic, phosphorus, &c.

Multiple neuritis in Beri-beri or Kak-ke, in syphilis, and in tuberculosis requires separate mention.

That multiple neuritis is the essential part of Beri-beri is extremely probable, but can hardly yet be held as proved. The disease is common in Japan, China, the Dutch colonies, and Brazil. It affects by preference young male adults, especially in the months of July–September, and when they are crowded together in prisons and barracks. Scheube[2] distinguishes four forms.

(*a.*) A slight one, characterised by great weakness of the legs, œdema, and palpitation of the heart: recovery is the rule after a few weeks or months.

(*b.*) An atrophic form, with weakness of the legs, proceeding to complete paralysis and muscular atrophy. The arm may be affected, and more rarely the tongue and face. Death from exhaustion is a not unfrequent result, or recovery after a very considerable time.

(*c.*) The dropsical form. (*d.*) The acute pernicious form.

The course of the disease presents great similarity with multiple neuritis and with Landry's paralysis. Bälz and Scheube found the peripheral nerves affected exactly as in multiple neuritis, and the spinal cord unaltered. They consider Beri-beri to be an infectious endemic, sub-acute, multiple neuritis.

Peckelhering and Winkler, who were appointed by the Dutch Government to investigate the disease, also express the opinion that it is an infectious multiple neuritis. They found the peripheral nerves degenerated, and they also found changes in the cardiac nerves. They state that they found rods and diplococci, which they consider to be the cause of the disease, and which, when injected into dogs and rabbits, produced a degeneration of the nerves.

Quite recently Dr. C. K. Mills, of Philadelphia, U.S.A., has described several cases of neuritis following, or rather occurring during, epidemic cerebro-spinal meningitis. Dr. Osler noticed as a peculiarity that, unlike other examples of this complication of specific fevers, the symptoms appeared early in the course of the disease. No pathological evidence was offered in confirmation of the diagnosis; but the disease affected specially the lower extremities, the knee-jerk was diminished, there was loss of power, the feet were in a position of equino-varus, there was soreness over the nerve trunks, with muscular and cutaneous hyperæsthesia—a group of symptoms pointing very strongly to neuritis as the cause.

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[1] "Zur Kenntniss der akuter infektiösen multiplen Neuritis:" 'Arch. f. Psych.,' xviii. 3.

[2] "Die japanische Kak-ke:" 'Deutsch. Arch. f. klin. Med.,' xxxi. u. xxxii.

With regard to multiple neuritis due to syphilis, Professor Leyden thinks that such an occurrence can no longer be doubted, although in the literature of the subject scarcely any mention of such cases is made.

Much the same may be said of tuberculosis as a cause of multiple neuritis. Professor Leyden, however, considers that there is no reason to suppose that the tubercle bacillus, either directly or by producing a ptomaine, is the cause of the neuritis, which he attributes rather to the phthisical cachexia.

Coming to the toxic form, Professor Leyden devotes most attention to lead paralysis as the most frequent and most important. Though for a long time it was supposed that the spinal cord was at fault, and that the cause of the disease was poliomyelitis, there can now be no doubt that lead palsy is essentially a neuritis. It differs from other forms of multiple neuritis not only in its peculiar localisation, but also in the relatively speedy oncoming of irreparable degeneration. In rare cases it affects the lower extremities also, and becomes more or less generalised. It then more closely resembles ordinary multiple neuritis. In such generalised cases degenerations of the cord of the nature of poliomyelitis are usually found in addition to the peripheral neuritis.

Of phosphorus neuritis but little is known. It appears to closely resemble arsenical neuritis, which has been recently described by Naunyn [1] and by Dana.[2] There seems to be special liability to the affection of sensory nerves.

Mercury, besides causing tremor, may give rise to neuritis. This form of paralysis has been recently studied by Letulle.[3] Experimental investigations also show that the affection of the nerves produced by mercury is degenerative and not inflammatory. Of the same nature is the paralysis and neuritis produced by the subcutaneous injection of ether, recently studied by Pitres and Vaillard, Mendel, and Remak.

Alcoholic neuritis has attracted much attention of late, and deservedly. Professor Leyden divides it into four classes:—

1. Alcoholic tremor, of the physiological cause of which nothing definite is known.

2. Alcoholic paraplegia. 3. Alcoholic ataxia.

4. The hyperæsthetic form due to chronic alcoholism.

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[1] 'Berl. klin. Wochenschrift,' 15, 8, 86.

[2] "On Pseudo-tabes from arsenical Poisoning, with a Consideration of arsenical Paralysis:" 'BRAIN,' 1887, p. 456.

[3] "Recherches cliniques et expérimentelles sur les Paralysies mercurielles:" 'Arch. de Physiol. norm. et Path.,' 1887. See 'BRAIN,' April 1888, p. 140.

With regard to the etiology he remarks that, in addition to the action of alcohol, most patients give a definite history also of exposure to cold, &c. In the ataxic form the sensory nerves are especially affected. In the hyperæsthetic form the pains are excessive and are not readily relieved. As in all forms of alcoholic neuritis, the lower extremities are most frequently affected. There may be in addition motor weakness and disturbances of sensation.

The spontaneous or primary form of multiple neuritis is attributed to exposure to cold or over-exertion, or both combined. It generally affects the lower extremities, sometimes the upper and occasionally both. It is frequently accompanied by fever.

The atrophic, anæmic, or cachectic form of multiple neuritis is the least known and has been the least studied of all, although it has long been noticed that various forms of paralysis occur during convalescence from severe illnesses, and in cases of profound exhaustion. At present pathological anatomy has not afforded any proof of the existence of neuritis in these cases, but the probability of such evidence being forthcoming in the near future is rendered very great by the interesting observations of Oppenheim and Siemerling.[1] These authors, while investigating the degeneration of the peripheral nerves in tabes, examined the condition of the nerves in a considerable number of other diseases. They found that in long-continued and exhausting diseases there is usually a very definite change in the peripheral nerves. Such changes were found in simple inanition, senile marasmus, cancer cachexia, alcoholism, and acute infectious diseases.

According to Professor Leyden, diabetic neuritis should be placed in the same category. It appears to bear no direct relation to the glycosuria. Though usually occurring in severe cases, it has been found where there was only ½ per cent. of sugar. It is not benefited when the proportion of sugar has been reduced by strictly limited diet: it may continue after the sugar has entirely disappeared, and may get well while the quantity of sugar remains unaltered.

The varieties of diabetic neuritis closely resemble those of chronic alcoholism, and may be divided into three classes: *a.* The hyperæsthetic. *b.* The motor. *c.* The ataxic.

And lastly we come to the sensory or ataxic form of neuritis, closely resembling tabes dorsalis, and therefore often called pseudo-tabes.

So far back as 1863 Professor Leyden, in his monograph 'On the Grey Degeneration of the posterior Columns of the Cord,' put forward the theory, that the disease consisted essentially in a gradually progressive degeneration and atrophy of the sensory tracts, including both the sensory columns of the cord and the sensory nerve-roots; and he attributed the ataxic symptoms to the loss of sensibility.

In 1878 Westphal published the result of his investigations into the degeneration of the peripheral sensory nerves in tabes.

In 1879 and 1880 Pierret showed that in tabes the spinal and cerebral sensory nerves, including the optic nerve, are frequently the seat of peripheral changes, in no way continuous with the central (spinal) affection.

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[1] "Beiträge zur Pathologie der Tabes dorsalis, und der peripheren Nerven Erkrankung:" 'Arch. f. Psych.,' xviii. 2.

More recently Oppenheim and Siemerling have found, in the majority of cases of typical tabes, marked alterations in the cutaneous nerves. The peripheral nature of the affection is shown by the fact, that the large nerve trunks are much less affected, and that the disease of the cord is in no relation with the degeneration of the peripheral nerves.

Déjérine[1] in 1883 attributed to sensory peripheral neuritis the sensory and trophic disturbances in the skin of tabetics.

Pitres and Vaillard,[2] in 1866 found degeneration more frequent in the smaller branches than in the nerve trunks, and affecting motor, mixed, and visceral nerves. They think that in tabes there is an acute or sub-acute neuritis which is capable of resolution.

All this has gradually paved the way for the recognition of a form of ataxia or pseudo-tabes dependent on a peripheral sensory neuritis, without any affection of the spinal cord or nerve-roots.

Déjérine,[3] who has done the most for the elucidation of this disease, describes two forms:—

a. The acute or sub-acute, often accompanied by motor affections also, and coming on after acute diseases, such as diphtheria, enteric fever, small-pox, &c.; and also in toxic conditions like lead and arsenic poisoning, and in diabetes.

b. The second form consists in a sclerotic atrophy of the peripheral sensory nerves, and is chronic in its course. It much more closely resembles true tabes. Both these forms of pseudo-tabes are capable of recovery more or less complete.

And now a very interesting and important question is raised. Can true tabes arise from a peripheral cause, and is it, at a certain stage of its development, when it already displays characteristic symptoms, limited to a peripheral change apart from any lesion of the cord? The practical importance of the question is this. Most forms of peripheral neuritis have been shown to be capable of recovery. If some forms of tabes commence as a peripheral neuritis they should in that early stage be curable, and the prognosis, when the diagnosis is made sufficiently early, will be less unfavourable.

And, finally, Professor Leyden asks another question: Can the acute ataxia of sensory peripheral neuritis pass into the chronic degenerative form, and then affect the cord secondarily, and so develop typical tabes? This must be left for future investigation.

H. Handford, M.D.

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[1] 'Arch. d. Physiologie et Pathologie,' 1883.

[2] "Contribution à l'Étude des Névrites périphériques chez les Tabétiques:" 'Revue de Médecine,' 1886.

[3] 'Compt. rend. de l'Acad.,' 1883

RECENT BOOKS ON PHYSICAL PSYCHOLOGY.

Grundzüge der Physiologischen Psychologie. Von WILHELM WUNDT, Professor an der Universität zu Leipzig. Dritte umgearbeitete Auflage. 2 Bände. Leipzig: W. Engelmann, 1887. pp. xii. 544; x. 562.

Eléments de Psychologie Physiologique. Par W. WUNDT. Traduits de l'Allemand sur la deuxième édition avec l'autorisation de l'auteur par le Dr. Elie Rouvier, de Pignan. 2 Tomes. Paris: T. Alcan, 1886. pp. xxxii. 571, 532.

La Psychologie Physiologique. Par G. SERGI, Professor d'Anthropologie à l'Université de Rome. Traduit de l'Italien par M. Mouton, Professeur de Philosophie au Collège d'Armentières. Edition française, revue, corrigée et augmentée par l'auteur. Paris: T. Alcan, 1888. pp. iii. 452.

Elements of Physiological Psychology. A Treatise of the Activities and Nature of the Mind from the Physical and Experimental Point of View. By GEORGE T. LADD, Professor of Philosophy in Yale University. London: Longmans, Green & Co. (New York: Charles Scribner's Sons), 1887. pp. v., 696.

The American Journal of Psychology. Edited by G. STANLEY HALL. Professor of Psychology and Pedagogics in the Johns Hopkins University. Vol. I. Nos. 1 and 2. Baltimore: N. Murray. 1887–8.

Psychologie in Umrissen auf Grundlage der Erfahrung. Von Dr. HARALD HÖFFDING, Professor an der Universität in Kopenhagen. Unter Mitwirkung des Verfassers nach der zweiten dänischen Auflage übersetzt von T. Bendixen, Gymnasiallehrer. Leipzig: Fues's Verlag (R. Reisland) 1887. pp. vi. 463.

Psychology. [James Ward]. Encyclopædia Britannica. Ninth Edition. Vol. xx. Edinburgh: Adam and Charles Black. 1886.

THESE writings bear witness to the increasing interest everywhere taken in physiological psychology. Germany, France, Italy, Scandinavia, and America are represented, and if we add to the list the Russian translation of Professor Wundt's work, and Mr. Mercier's 'The Nervous System and the Mind,' we find a forward psychological movement in all the countries which show scientific activity. It is by no means improbable that the time has now come when the most careful research and the best thought will be given to the investigation of mind and its relation to matter. When methods of exact observation and experiment were first used, they were naturally applied to the physical world. To begin with, it was found easier to study the constant phenomena of dead matter than the ever-changing phenomena of the living organism, and physics, including astronomy and chemistry, were well advanced, before biology and physiology had become exact sciences. Physics culminated with the doctrine of the conservation of energy and biology with the doctrine of the evolution of species; and these sciences are at present largely engaged in working out details. Now that we have made such advances in physical science, and have learned from it correct methods of investigation, it would seem to be time to take up psychology, the phenomena of which are beyond all others complex and transient. It has even been claimed that mind is altogether beyond the reach of scientific investigation, but the books given above prove that this is not the case, and the immense importance of the subject will lead to strenuous effort.

Professor Wundt's masterly 'Physiologische Psychologie' deserves to stand at the head of our list. In this third edition the work has been throughout revised, and its size increased by 145 pages. The table of contents is, however, substantially the same as in the preceding edition, the subjects of the sections being as follows: Vol. I., Introduction (1–20); I. The Physical Basis of Mental Life (21–228); II. Sensation (289–544). Vol. II., III. The Formation of Presentations (1–224); IV. Consciousness and the Train of Ideas (225–462); V. Volition and Volitional Movements (463–530); VI. The Genesis of Mental Development (531–554); Index (555–562). The work covers so much ground, and in a manner so thorough, that it is impossible for me to review the contents of the several chapters and the changes in this edition. Readers of 'BRAIN' will, perhaps, be especially interested in the first section. Here the anatomy and physiology of the nervous system were discussed with great thoroughness, and with reference to the most recent researches; for example, Professor Meynert's 'Psychiatrie,' and the second edition of Dr. Ferrier's 'Functions of the Brain.' But while the subjects treated in this section are those with which 'BRAIN' is most nearly concerned, its readers may, perhaps, learn more from the following sections. Many will be surprised to note the rapid progress lately made by experimental psychology, or psychophysics, not by study of the nervous system, but through investigation of mind itself. Introspection has an ill name among neurologists, and deserves it in so far as it denotes the isolating of such half-observed mental phenomena as will fit into some preconceived theory. But the orderliness of mind is no less than that of the rest of nature; it can be studied by correct methods of experiment and observation, and we may hope that in the end psychology will become a science as exact as physics. Indeed, even now psychology is in many respects far in advance of neurology; when we perceive or think, we know much better what happens in consciousness than what happens in the body. Thus theories such as those of Helmholtz and Herring, to explain the process concerned in vision, go from observed mental facts to doubtful physiological assumptions. Any one who wishes to learn what is known concerning sensation and perception, mental intensity and time, thought, feeling and volition, will find it all set forth with admirable order and completeness in Professor Wundt's 'Physiologische Psychologie.'

Unfortunately the French translation of Professor Wundt's work is from the second edition, and is now to a certain extent obsolete. It may, however, be of use to those who do not know German. The translating seems to be accurately and well done. An English version of the third edition would be extremely welcome and useful.

In the case of Professor Sergi's work, the French translation has been revised and augmented by the Author, and may be looked upon as a new edition of the Italian original published in 1879. The book covers the same ground as Professor Wundt's, but is not nearly so well arranged. It may be recommended to the advanced student, but will scarcely be profitable for those who do not already know both physiology and psychology.

Professor Ladd's book, on the other hand, will prove most useful to those whose training has been exclusively philosophical or physiological. We may rejoice that we now have in the English language an elementary, but at the same time tolerably complete treatise on physiological psychology. After a short Introduction (1–14), the book falls into three parts: I. The Nervous Mechanism (17–236); II. Correlations of the Nervous Mechanism and the Mind (239–582); III. The Nature of Mind (585–688). Readers of 'BRAIN' will already know the contents of the first part, as they are a summary of the familiar facts of neurology. The second part will, however, be found new and interesting by many. The several chapters treat (after two on "The Localisation of Cerebral Function") "The Quantity of Sensations," "The Presentations of Sense," "The Time-relations of Mental Phenomena," "Feelings and Motions," "The Physical Basis of the Higher Faculties," and "Certain Statical Relations of the Body and Mental Phenomena." In the third part Professor Ladd seems to give up much of the ground gained in the first and second parts. He argues that mind is "a real unit being," standing in causal relations with the brain.

Professor Ladd's book comes from America, and another sign of psychological activity in that country is the new Quarterly edited by Professor G. Stanley Hall, and published by the Johns Hopkins University. From the prospectus and the contents of the first three numbers we may judge that the journal will cover much the same ground as 'BRAIN.' Some of the subjects discussed are "The Knee-Jerk," "Dermal Sensitiveness," "The Horopter," "The Psycho-physic Law," "Neurology and Psychology," "Fixed Ideas," "Psycho-physic Methods," "Dreams," "Paranoia," "Stimulation of Ganglion Cells," &c. Each number contains also extended Book Reviews and Notes. It is announced that Professor Hall will leave the Johns Hopkins University, so there is room for doubt as to what is to be the future of the journal.

The two works last on the list do not, strictly speaking, come under the heading "Physiological Psychology," but the authors show thorough acquaintance with the subject, and make use of it so far as a treatise on psychology requires. Professor Höffding's book is an excellent introduction to psychology, clear and complete. Dr. Ward's article is more difficult reading, too advanced and condensed, perhaps, for an Encyclopædia. But it is the most important contribution recently made to psychological science.

J. McK. Cattell.

Dissolution and Evolution and the Science of Medicine.—By C. Pitfield Mitchell, M.R.C.S. (Longmans, Green & Co., 1888, pp. 246).

Herbert Spencer, in his 'First Principles,' conceives that "Evolution is definable as a change from an incoherent homogeneity to a coherent heterogeneity, accompanying the dissipation of motion and the integration of matter." This he expresses in the form of a formula, thus:—

Evolution is an integration of matter and concomitant dissipation of motion; during which the matter passes from an indefinite, incoherent homogeneity to a definite, coherent heterogeneity; and during which the retained motion undergoes a parallel transformation.

Mr. Pitfield Mitchell has imposed upon himself the task of applying this formula to many of the leading facts of pathology in its relation to medicine. Every one will of course agree with him that "to make all diseases, from a whitlow to mania, one in principle by cause and effect, is an aid to practical thought," but it is certainly a stupendous mental undertaking to endeavour to bring about such a desirable state of affairs. We shall now see how far the treatise under consideration tends to accomplish this.

We learn from Spencer that, "A history of anything must include its appearance out of the imperceptible, and its disappearance into the imperceptible."[1] Mr. Mitchell reverses this order, and begins by considering the disappearance into the imperceptible.

Chapter I. is concerned with Inflammation and Suppuration as dissolutional changes; unfortunately the author does not present his views in a clear and lucid manner, nor is the description of the inflammatory process adopted by him to be found in text-books which he tells us in the Preface are his authorities. Hence, if the data are loosely given, we must of necessity regard the conclusions drawn from them with grave suspicion.

Chapter II. deals with Resolution and Repair as Evolutional changes. These are held up by Mr. Mitchell to illustrate "Integration of matter and dissipation of motion;" but the imperfection of our knowledge concerning the fate of inflammatory products undergoing resolution, makes one regard the views expressed in this chapter as very visionary.

Chapter III. is devoted to Retrograde metamorphoses as dissolutional changes. It consists chiefly of a very superficial review of what is known regarding coagulation of the blood, thrombosis, fatty degeneration, and the like. Most of these changes are admitted by pathologists as of the nature of dissolutional processes.

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[1] 'First Principles,' chap. xii.

Chapter IV. is very short, but contains some odd opinions as to the changes induced by vegetable and animal parasites.

Chapter V. is a very long one, dealing with Neoplasms as exemplifying dissolution. It contains, besides a new theory of tumours, many singular statements unsupported by facts. Cohnheim's theory of embryonic rudiments is rejected, an endeavour being made to supplant it by regarding the formation of a tumour as "a 'seedling' of the natural plastids of the body," and that it is "a process of agamogenesis, or asexual reproduction known in Protozoa, and in all probability *is a reversion to this primal type of multiplication.* For the arguments in support of this we must refer the reader to the book itself, but we are bound to confess that Mr. Mitchell's does not convince us of the probability of this theory, and he uses the terms tumour, cancer, cyst, neoplasm, &c., in such an indefinite manner as to prevent the reader grasping the meaning.

On page 107 mention is made of the fact, "that congenital cystic kidneys are now thought by some observers to depend upon the degeneration of persistent fœtal" (in the book it is misprinted fatal) "structures, *e.g.* the Wolffian bodies"; but as there are two forms of congenital cystic kidney, we are left in doubt to which the author refers, especially as he fails, as is so often the case, to make any reference to the original observations.

The second division of the book deals with special diseases, as exemplifying dissolution and evolution. It is very unfortunate that the various forms of Bright's disease should have been selected to illustrate the principle, for any hypothesis based on such ground as the pathology of renal changes, resembles a house built on sand. This applies with equal force to locomotor ataxia, a useful label for an interesting but variable group of symptoms with an inexact pathology. The same may be said of the remarks on the pathology of diabetes.

In diseases of the mind, with no pathological facts to control the hypothesis-maker, fancy may take wings and unrestrainedly defy the critic; it is so in this book.

Part III. is headed Implications, and contains a summary of the preceding chapters; but fails to render them clearer. A section is also devoted to Heredity and Disease, and one follows on Equilibration in Disease, containing some very singular opinions; finally the book is ended by the consideration of some practical deductions and conclusions, chiefly, a repetition of well-known facts. It is much to be regretted that a study, which ought to have been fascinating and interesting, should fail because the writer adopts an ambiguous and pedantic style, is not over-cautious in utilising facts, and is careless in quoting descriptions of well-known experiments. In a difficult task like the one attempted by Mr. Mitchell, lucid explanations of difficult processes are essential to success.

J. Bland Sutton.

Abstracts of British and Foreign Journals.

Oppenheim on Cerebro-Spinal Concussion (*Berlin. Klin. Woch.* No. 9, S. 166, 1888).—The author commences an interesting paper by indicating the change that has taken place in the nosological conception of the nervous diseases induced by concussion. In earlier times, it was assumed that the traumatic force of a concussion, such as may ensue from a railway accident, was chiefly directed to the spinal cord, and under the influence of that view those symptoms which could with some probability be referred to a disease of the cord were mainly considered. Since then certain brain symptoms, which though not entirely overlooked were regarded as casual or unessential, have received keener scrutiny and consideration. Railway-spine (the old term used for denoting these cases) has grown into Railway-brain, traumatic meningo-myelitis into traumatic hysteria.

As the basis for a more accurate classification and nomenclature, the author details the results of his extensive clinical experience of such cases.

The symptomatology of these morbid states is exceedingly manifold and diverse. In the greater number of cases the most essential alteration involves the mind, especially the affective sphere. Melancholy and excitability form the core of the psychosis. The patients are gloomy, immersed in painful ideas, reticent; they prefer solitude. This depression is accompanied by a feeling of fear, which at times rises to violent paroxysms of terror. Although there is no delusion in the strict sense of the word, the recollection of the accident is so vivid, so largely determines the thoughts and feelings of the patient, that it attains a pathological character by its constancy and dominance in the mind. It prevents sleep, or influences the tenor of the dreams, so that the patient whimpers, shrieks, starts up, or springs from the bed in a state of frenzy.

There are two features which, in the author's opinion, differentiate this mental alteration from pure melancholia, with which it has at least a great affinity in most cases, viz. the abnormal irritability and the decidedly hypochondriacal character of the mental disturbance. These persons, though brooding over their condition and absorbed in themselves, still are morbidly sensitive to external irritants. They shrink at slight noises, are thrown into agitation by circumstances which formerly would have failed to move them, are suspicious and easily mortified, readily burst into tears, and suffer themselves to be carried away by their feelings. Moreover, being swayed by the idea that the accident has caused them to be profoundly ill, they watch for, and exaggerate, the slightest disagreeable sensation.

Severe disorder of the intellect is seldom present, yet considerable dementia with progressive enfeeblement of memory sometimes occurs. Mostly one obtains only the impression that the patients, influenced by depressing emotions and the consciousness of disease, have not free exercise of their mental powers. Many of the patients easily become perplexed and complain of great absence, so that they may even lose their way in the streets, or enter the wrong house, &c.

Hallucinatory delirium at times accompanies the paroxysms of fear before mentioned.

The psychical anomaliés may assume the form of traumatic insanity, rendering asylum control necessary in a small proportion of cases. Agoraphobia and acrotephobia have been observed.

Vertigo and spasms—the latter always belonging to the category of hysteric convulsions—are frequent. The vertiginous attacks may be attended with loss of consciousness, or prolonged hystero-epileptic seizures may occur.

Sensory disorders are nearly always present, though in manifold variety and intensity. They mostly show the seeming anomaly that characterises the mental affection—hyperæsthesia and anæsthesia are found running side by side in the same receptive sphere. Scintillæ and great sensitiveness to light coexist with a diminution of visual acuity, mainly in the form of concentric contraction of the field of vision. Dysacusis and tinnitus are present with a demonstrable enfeeblement of condition. Frequently a great part of the cutaneous surface is insensitive, while in the anæsthetic region there are hyperæsthetic zones; or there exists a hypersensitiveness to slight pressure or contact, with analgesia to severe stimuli—*e.g.* transfixion of a fold of skin.

Of much import in connection with this anæsthesia are the facts, that it never coincides with any particular nerve territory, and it most rarely has a distribution such as occurs in spinal cord disease. Often there is merely a tract along the outer aspect of the lower extremity.

There are cases in which only a single limb or portion of a limb was affected by the trauma, and subsequently a total hemianæsthesia of the same side has developed. Girdle pain is seldom complained of.

Sometimes the muscular sense is impaired to such a degree, that the patient requires to control all movements with the eyes.

Dull, compressive, non-lancinating, dorsal pains are common, and lead to arrest of voluntary movements. Headache is frequent.

Myotatic irritability usually is increased. The superficial reflexes may be exaggerated, more often they are lessened.

Disturbances of motility are almost always demonstrable. Movements are executed slowly and without energy. One may often ascertain that, if the patient exert stronger volition, there is a reserve of motor capacity.

Very diverse are the abnormalities of gait; they do not usually agree with those appearing in material diseases of the spinal cord, and doubtless in this matter a psychical element frequently plays a part. Sometimes the peculiarity is of such a nature as to suggest malingering. One patient could walk backwards with greater ease than forwards. In the rare forms of unilateral palsy the affected leg trails, but in a manner which deviates in certain particulars from the dragging observed in ordinary hemiplegia. Reeling, on closure of the eyes, is often to be noticed.

Tremor is very common: it is specially apparent in the extended hands and usually has the characters of that occurring in hysteria, neurasthenia and alcoholism. The tremor movements may be so violent that the whole body is agitated. It is notably increased by mental excitement: rarely is it limited to voluntary efforts.

Speech suffers in many cases; not in the form of a genuine aphasia, bulbar paralysis or scanning utterance: the anomalies are peculiar—the patient breaks off in the middle of a sentence as if he lost the thread; or single words are emitted tardily, others explosively; or the speech is interrupted by frequent inspirations and sobs. Some patients explained their peculiarity of speech by confessing, that they forgot in the middle of a sentence what they wished to say.

Attacks of palpitation, combined with fear, quickening of the pulse, and sometimes with flushing of the face, dilated or unequal pupils and tremor, are frequent symptoms. In other cases there may be constant acceleration of the pulse, or blushing of the face, neck and breast, with axillary hyperidrosis, may occur. Dilatation and hypertrophy of both ventricles developed as a consequence of the cardiac disturbance in one patient.

In a large number of cases the general nutrition suffers: anorexia, constipation, impotence may be present. Vomiting, thirst, polyuria, and pyrexia appeared in two of the patients. Disorder of the bladder function is rare. Sluggishness and inequality of the pupils are frequent. Reflex iridoplegia existed in only a small fraction of cases, viz. five. Optic-nerve atrophy was found in only two patients.

Analysis of the above sketched clinical phenomena must lead to a conception of these morbid states which differs essentially from those formerly prevailing. The old theory, that a meningo-myelitis develops in consequence of the injury, received the assent of German authors, and the term Railway-spine—the product of that theory—was accepted by them. Later, the French school, perceiving the analogous nature of the sensory troubles in railway cases and hysteria, has proclaimed the diseases of the nervous system due to railway accidents to be nothing but hysteria. Charcot further showed that during hypnotism paralytic conditions of the same character as those observed after accidents, and accompanied by the same forms of sensory disorder, can be produced by suggestion or by a slight blow upon the extremity.

But it is seldom a pure psychosis, such as melancholia, hypochondriasis, simple dementia; or a pure neurosis, such as epilepsy, hysteria, neurasthenia, which results from the injury: usually the form of disease is mixed and anomalous. At any rate the essential symptoms point to a cerebral basis, and, though it is not denied that a local lesion of the spinal region may sometimes form the starting-point of the affection (reflex neurosis), the term Railway-spine is quite inappropriate for the majority of cases. Neither does it suffice to substitute the nomenclature, traumatic hysteria or traumatic neurasthenia: the true classification places the greater number of cases under traumatic neurosis, or traumatic neuropsychosis.

Obviously the material changes giving rise to severe vesical troubles, reflex pupil rigidity, or optic-nerve atrophy, would exclude the few cases in which these signs occur from the above categories.

In general the life of the patients does not seem to be shortened, but the prognosis quoad recovery is altogether bad—the author has not been able to ascertain one case of restoration to health.

The worries associated with litigation notably increase all the troubles. After solution of the compensation question, amelioration often is observed. The diagnosis between disease and malingering may require prolonged observation, but usually is not difficult. Morbid states, exactly similar to those above described, originate from concussion due to other causes than railway accidents—*e.g.* machinery accidents in factories—where the question of indemnification is entirely absent.

Regarding the antecedents of the author's patients, hereditary predisposition to nervous disease could very seldom be made out. Alcoholism was more frequent.

Pomorski on Plexiform Neuroma of the Intercostal Nerves, with Fibroma Molluscum and Neurofibromata (*Virchow's Archiv*, January, 1888).—The subject of this rare combination of morbid developments was a strongly-built corpulent man, aged 28. Dyspnœa and severe pain in the right side of chest, which had existed a few weeks, occasioned his entrance into hospital.

The clinical diagnosis was:—Sarcoma in right side of thorax, Pleuritis hæmorrhagica, Ascites, Fibroma Molluscum, Hæmophilia. Death occurred in three weeks. On the skin of the trunk and extremities there were numerous warts, varying in diameter from ¼ to 5 cm., none exceeding ¾ cm. in height; most were sessile, few pedunculated. Nearly all presented a rough wrinkled surface composed of thickened dry epidermis: deep groovings and constrictions of the larger ones indicated their formation by the coalescence of many small nodes.

Right lung entirely collapsed. From the clavicle to the fourth rib the right pleural cavity was filled by two globular fibrous tumours, each as large as a child's head: their surface was smooth, light grey or yellowish, glistening and gelatinous in appearance; consistence, tense and elastic. Concealed by these tumours was a large mass of convoluted, ramose, tuberous varicose cords, spreading outwards from the vertebral column over the costal pleura, united to each other and to contiguous parts by a fine soft, loose, connective. Some coursed in serpentine form to the under surface of the fibromata. At the one end a cord was bulbous; at the other it was found to spring from an intercostal nerve by from one to three fine processes, which ran spirally through the connective tissue. The cords generally were firm, but here and there soft and gelatinous; where firm, the appearance was fibrous; in other parts, yellow and shining.

At the vertebral column the interstitial tissue was very thick, and formed pouches, with smooth walls, surrounding the cords, and the latter had grown through the intercostal space to the outer surface of the ribs, and lay enveloped in a layer of dense connective tissue, which adhered to the thickened periosteum.

Projecting from the surface of costal pleura were red, soft nodules, the size of a lentil or pea, scattered over both ribs and interspaces.

The intercostal nerves were thickened to about double their normal size, and showed numerous fusiform and nodular swellings, giving rise to a rosary-like appearance. The nodules in pleura were most closely set where this appearance was most pronounced.

The trunk and branches of the nerves of upper limbs, also the vagi, exhibited considerable general thickening with multiple fusiform nodes, and excentrically placed gelatinous spindles. The smaller branches of the brachial nerves, especially, had a varicose or rosary-like contour. Accurate dissection failed to show that the node formation in the nerves exactly corresponded to the multiple nodes in the skin, or that the terminal branches of the nerves entered the molluscal nodules; but a close relationship between the three forms of tumour was evident. The fibroma molluscum consisted of small growths of yellowish, soft, gelatinous connective tissue; similar tissue formed the enlargements of the nerves, and the substance uniting the cords of the plexiform neuroma, as well as the membrane investing the large fibromata.

Respecting the histological features:—In the fibroma molluscum, long cones of epithelium were seen growing into the tumour mass from the thickened Rete Malpighii. In the obliterated hair follicles, without evidence of a preformed canal, lay globules of concentrically disposed epithelium, having a dull silvery lustre. The tissue beneath the epidermis consisted of a delicate wide-meshed reticulum, formed solely by the processes of cells, with a soft homogeneous ground-substance in the meshes. Small round and fusiform cells existed on the reticulum. At the limit of the corium and subcutis were large and small soft fibrous nodes, formed by small round cells, with large nucleus, densely crowded together and surrounding sweat-coils and ducts, vessels and hairs. The cells in the larger nodules were mainly spindle shaped. In almost every small nodule a sweat-coil or the duct of a sudiparous gland could be seen. The elongation of, and accumulation of glistening yellow hyaline contents in, the canals, as observed by Recklinghausen, were present in this case. The sweat-ducts were surrounded by a thick layer of round cells by which the connective tissue-sheath was almost completely destroyed—the still visible epithelium forming an axial cord of an elongated body.

Next to the sheath of the sweat-glands the sheaths of the vessels partook in the formation of the fibrous nodules, by hyperplasia of their connective tissue: a thick fibrous adventitia was most perfectly seen. The part taken by the hair follicles was considerable; many of them showed cell proliferation in their connective tissue-sheath, and were encircled by fibrous tissue. Nerves were seldom seen in the nodules. In one section a primitive nerve fibre entered a small node laterally; in another, three medullated primitive fibres, lying parallel to each other, ran to the under edge of the tumour.

Sebaceous glands seemed to take no part in the new formations. Along the delicate fibres of the reticulum cells were accumulated in small heaps or longish strands.

Longitudinal section of a large node of the median nerve showed an excentric gelatinous nodule which was quasi-encapsuled, and could be easily shelled out. It was composed of a soft, juicy, connective tissue, provided with many round and spindle cells, with homogeneous intercellular substance and scanty short fibrillæ. The nerve fasciculi were pressed aside and flattened by the nodule, but exhibited normal amount of myeline substance. One bundle traversed the nodule—its primitive fibres being dissociated in a radiate manner by the proliferous endoneurium. At the point of entrance into the nodule, the nerve fibres were stained deep black by perosmic acid; but within the nodule they were the paler the more widely they were separated, evidently from atrophy of their medullary sheath. In one series of cross-sections made below the node, hyperplasia of the inter- and intrafascicular connective tissue, with corresponding wasting of the medullary sheath, was seen. Wherever the endoneurium was normal, the nerve fibres were sharply and clearly stained. By the use of carmine after treatment with osmic acid, one saw, on cross section, in many places where normal medullated nerve tubes were to be expected, small roundish bodies composed of a limiting membrane, three to five deeply stained nuclei, and clear glistening non-colourable substance. These were not regarded as being due to a division of the axis-cylinder, but to a nuclear proliferation in the nerve sheath, since longitudinal section disclosed no axis cylinder, only groups of small oval nuclei.

In the slender processes connecting the varicose cords of the plexiform neuroma with the intercostal nerves, nodulated nerve fibres, medullated and grey, were present ensheathed by a soft cellular and vascular connective tissue. In the cords themselves were closely interlaced twisted fibres, having a vitreous homogeneous appearance, which stained light yellow with picro-lithiocarmine. On longitudinal section the centre stained rather more deeply with carmine than the periphery. In a transverse section one saw a sharply stained circular centre surrounded by glistening unstained material and a single contoured sheath.

ERNEST BIRT.

Martius on Tabes Dorsalis, with Presence of Knee-jerk and Absence of other Symptoms (*Deutsch. Med. Woch.*, March 1st, 1888).—The author describes a case interesting on account of the absence of the usual diagnostic symptoms, and the degeneration subsequently found in the spinal cord.

The patient was a man, aged 53, who was affected with spinal symptoms after an attack of typhoid fever. He first suffered with paræsthesia, limited exclusively to the upper extremities; the hands and tips of the fingers being numb and stiff. The same condition appeared later in the lower extremities, followed by weakness of the lower limbs. When he came to the hospital, five months after the first symptoms showed themselves, he complained of numbness in the hands and feet, and weakness in the knees. Knee-jerk present both sides, no Argyll-Robertson pupils, and he could stand with his eyes shut, thus the principal symptoms of ataxy were absent. The single sensory symptom was, patient could not bring his fingers together with the eyes closed, and felt as if there were thick gloves on his hands. About three months later, patient was seized with pneumonia, and died. Up to his death the knee-jerks were present on both sides. Examination of sections of the cord, after hardening in bichromate of ammonia, exhibited degenerations which quite agreed with the clinical symptoms presented during life. Dr. Martius compares the degenerations found with those artificially produced in animals by division of the posterior roots in various regions of the cord (Kahler and Singer). In the sections of the cord examined by him, he found absence of degeneration in the lumbar region, in the dorsal region slight degeneration of Goll's column, marked degeneration of the postero-external column surrounding it, but not of root zone. In the cervical region, Goll's column was only partially degenerated, but there was a very marked degeneration of the postero-external column, between the root zone and Goll's column.

As Professor Leyden pointed out in the discussion that followed, this singular condition could be explained if one considered that the degenerative process commenced in the posterior roots. The fibres which form the posterior median column run from the posterior roots into the postero-external column, then upwards and inwards towards the middle line. If then the posterior roots in the cervical region be affected, the degeneration would appear marked in the postero-external column of that region. A great number of the fibres which make up Goll's column come from the lower part of the body, and the clinical symptoms in this case showed that the lower extremities were only slightly affected; therefore it would not be expected that the posterior median column would be affected markedly in the cervical region, because those fibres which come from the upper extremity pass upwards and inwards from the postero-external column into the median column.

Pitres on Pseudo-Tabes (*Archives de Neurologie*, May 1888).—Patient was a strong and vigorous man, aged 35, previously having enjoyed good health. He was seized, without known cause, by various symptoms, analogous to those which characterise progressive locomotor ataxy; lightning-pains, motor inco-ordination of the lower limbs, gastric crises, &c. These symptoms persisted for nearly ten years. The patient died from an acute and intercurrent tubercular affection.

At his autopsy, no organic change was discovered in the spinal cord, the spinal roots, or peripheral nerves.

A very careful examination of a great number of peripheral nerves in different parts of the body was made, without discovering an abnormal change in any of them.

Among the visceral nerves submitted to histological examination were the great splanchnics, the pneumogastric, the superior laryngeal, right and left, the right recurrent laryngeal, and the right phrenic. In all the cases, the nerves were found to be quite healthy. The left recurrent nerve, however, showed considerable atrophic changes. The nerves of the stomach and of the heart also presented similar changes, no myelogenic fibres being visible.

The two principal diagnostic symptoms of tabes, namely, absence of knee-jerks and Argyll-Robertson pupils, were in this case wanting, and it is a question whether it is legitimate to make a diagnosis of locomotor ataxy in such a case. But still, as M. Pitres says, it is rare to find all the known tabetic symptoms present in one patient.

In the case under discussion, the patient had many of the symptoms which are so characteristic of locomotor ataxy, viz. lightning pains, gastric crises, and inco-ordination, without any atrophic affection of the muscles. The cutaneous sensibility was obtuse, the superficial reflexes were present, and the knee-jerks were normal.

M. Pitres concludes, that in certain cases functional troubles, susceptible of giving rise to a group of symptoms analogous to locomotor ataxy, and which would be diagnosed as such, may occasionally occur, as in this case; and he cites certain other instances in which a diagnosis of organic disease has been made during life by very competent authorities, but in which, at the autopsy, no organic change was discovered. He says, nothing is more difficult than to determine sometimes whether a nervous symptom is due to a pre-existing organic lesion, or if it is the result of a simple functional trouble. Symptoms which appear to indicate organic lesions, such as hemiplegia, hemianæsthesia, hemiathetosis, Jacksonian epilepsy, &c., are, in a good number of cases, independent of all organic changes in the central nervous system.

These two cases of Dr. Martius and Dr. Pitres are very instructive in connection with the diagnosis of tabes. In the former, we find that the disease affected principally the cord in its upper portion, hence the knee-jerk was present. In the latter, the knee-jerk was also present, but many of the symptoms pointed undoubtedly to locomotor ataxy. Dr. Pitres makes no mention of the condition of Clarke's column, and the fine nerve plexus in which the cells lie, but if there had been any change in either, probably it would have been noted. Still, the marked gastric crises would suggest the possibility that these cells might have suffered.

FREDK. W. MOTT, M.D.

Current Nerve Anatomy and Physiology.—Titles and Indication of Contents of Papers which have appeared during the first Quarter, 1888. By ALEX. HILL, M.D., Master of Downing College, Cambridge.

HUMAN BRAIN.

1. The Structure of the Central Nervous System in Health and Disease. OBERSTEINER (*Toeplitz and Deuticke; Leipsic and Vienna*, 1888).

An illustrated text-book of the anatomy, morphology, and histology of the central nervous system.

2. Anatomical Observations on the Brains of Criminals. MINGAZZINI (*Arch. Ital. de Biol.* ix., i., p. 31).

COMPARATIVE ANATOMY.

3. The Brain of the Seal (*Phoca vitulina*). THEODOR (*Berichte der Naturf. Gesellsch. zu Freiburg III.*, 1. p. 71, pl. viii.–x. 1888; also reprint, *Mohr*, *Freiburg i. Breisgau*).

Gives an account of the gross anatomy of the brain as seen from the surface, and with the help of such dissection as is necessary to expose the hippocampus, corpora quadrigemina, &c. Author says that the hemispheres are united together by two thick columns lying above the corpus callosum, for which he proposes the name commissura suprema.

Tuber cinereum is a small, sausage-like elevation, with a short longitudinal split. It shows no corpora mammillaria.

4. Anatomy of the Frog's Brain. KÖPPEN (*Archiv f. Anat. u. Physiol.*, *Anat. Abth.*, pt. 1, *March* 20, 1888, pp. 1–35, pl. i.–iii.).

An attempt, by means of sections stained according to Weigert's method, to distinguish the several bundles of fibres which enter into the formation of spinal cord, medulla, cerebellum, optic lobe, 'tween-brain and great brain, and trace their continuity.

5. Anatomy of the Frog's Brain. KÖPPEN (*Centralbl. f. Neurologie*, *Jan.* 1, p. 10, 1888).

Preliminary report of studies made with Weigert's method, and also with carmine-stained paraffin series. Observation of the position of nuclei in the medulla oblongata, particularly of vagus acusticus and trigeminus. The large vagus fibres which go to the semicircular canals are traced to large cells belonging to the column which elsewhere gives origin to anterior roots of spinal nerves. No fibre-column corresponding to the pyramidal tract could be found; the fibres from the cord reach no higher than the mid brain; fresh fibres connect the optic lobe with the cerebrum.

6. The Brain and Spinal Nerves of *Acipenser ruthenus* and *Amia calva*. GORONOWITSCH (*Morphol. Jahrb.*, xiii., pt. 3, pp. 427–514, pl. xvii.–xx., *March*, 1888).

A detailed account of the anatomy of these brains, both in the adult and fœtal fish.

7. The Classification of Gasteropods, based upon the Disposition of the Nervous System. LACAZE-DUTHIERS (*Comptes Rendus Acad. des Sciences*, cvi. No. 11, p. 716, *March* 1888).

MORPHOLOGY.

8. The Morphological Value of Cranial Nerves. HIS (*Arch. f. Anat. u. Physiol., Anat. Abth.*, 1887, pt. 6, pp. 378–453, issued *Feb.* 1888).

A critical study, based upon the paper of Gegenbaur, of which an extended account was given in these Abstracts in January last. (cf. 'BRAIN,' Vol. X., pp. 509–565.)

PHYSIOLOGY.

9. Concentration of Blood as a condition of Stimulus for the Central Nervous System. NOVI (*Lo Sperimentale*, pt. 5, 1887).

Injection of 10 per cent. salt-solution into the veins induces spasm of all the muscles of the body so soon as the concentration of the blood reaches double the normal. Not due to the salt-solution changing hæmoglobin into methæmoglobin. Not due to direct stimulation of the muscles, since the contractions are prevented by curare. Nor are the spasms due to action of the salt-solution upon peripheral nerves. They depend entirely upon stimulation of the brain, and are prevented by section of the medulla oblongata. These experiments would appear to illustrate the cause of spasms in diseases in which, as in Asiatic cholera, the blood becomes abnormally concentrated.

LOCALISATION.

10. The Functions of the Cerebrum (*Sixth communication*). GOLTZ (*Pfluger's Archiv*, xlii. pt. 9, pp. 419–467, pl. ii., *March* 1888).

A description of certain recent experiments of the brains, with illustrations, in photo-engraving. A dog was exhibited at Strassburg in June last, from which, by three successive operations performed in December 1885, and February and March 1886, the whole of the left side of the great brain had been removed. Detailed description of the animal's condition. It was able to move voluntarily all its muscles, although it preferred to move the left foot and side of the body rather than the right. Sensation on the right side was blunted. Vision disturbed.

The animal's intelligence was lowered. This experiment typical of several others.

Symmetrical removal of anterior regions and posterior regions with results, in the main, similar to those already recorded, confirming the author in the opinions expressed in his several papers (cf. *Pflüger's Archiv*, vol. xxxiv. p. 450, &c.).

11. Unipolar Induced Currents used for Stimulating the Brain. NEGRO (*Archives Ital. de Biol.*, ix. p. 48).

12. Physiology of the Motor-Area in the Dog. BECHTEREW (in Russian in *Archiv Neurologie*, 1887; Abs. in *Neurolog. Centralbl.*, *Jan.* 1, p. 14, 1888).

13. Experiments upon the Functions of the Cerebral Cortex. HORSLEY and SCHAFER (*Trans. Roy. Soc.*, vol. 179, B, pp. 1–45, pl. i.–vii., *April* 1888).

These experiments upon monkeys extend over a period of three years, and have reference especially to the centres for voluntary action and sensation. Gyri marginalis et fornicatus investigated for the first time. Excitation by Du Bois-Reymond inductorium and current not too strong to produce slight pricking sensation on the tongue. Ablation by means of the knife. Strictest antiseptic precautions. Regions investigated: I. prefrontal; absolutely negative results; electrically inexcitable area removed without loss of motion, sensation, or intelligence. II. Motor area mapped out into main territories and centres for specialised movements. Marginal convolution divided from before backwards into areas for movements; (1) of the head; (2) of the forearm and hand; (3) of the arm at the shoulder; (4) of the upper part of the trunk; (5) of the lower part of the trunk; (6) of the leg at the hip; (7) of the leg at the knee; (8) of the foot and toes.

III. Occipital lobes and angular gyri are concerned with visual perceptions, each occipital region being connected with the corresponding lateral half of each retina, a part of the cortex being able to take on in great measure the functions of the whole. Permanent hemiopia is produced when the removal of the occipital lobe is followed by removal of angular gyrus.

IV. Gyri fornicatus et hippocampi form a functionally continuous area, removal of parts of which affects the perception of cutaneous sensations on the opposite sides of the body. In some cases partial ablation was followed by localised loss of sensation. The paper concludes with a detailed account of forty-three experiments, illustrated by a great number of photo-lithographic plates.

LOCALISATION—THERMOTAXY.

14. The Question of the Localisation in the Brain of Centres for regulating Temperature, and the Action of Antipyrin on the Organism. SAWADOWSKI (*Medicin. Centralbl.*, Nos. 8, 9, 10, pp. 146, 162, 178; 1888.)

OPTIC LOBES.

15. The Inhibition Centres for Toads. ALBERTONI (*Centralbl. für Physiologie.*, No. 26*a*, *March* 17, 1888).

Author quotes from his former publication experiments which show that, while toads cannot be induced to relinquish the act of coitus by the very strongest sensory stimuli, the act is abandoned when the optic lobes are stimulated. From this he concludes that, for certain actions, the optic lobes function as inhibitory centres. Attention is called to these results as having been published before the papers of Tarchanoff abstracted in this Journal.

16. Experiments on the Inhibitory Nervous Centres of the Frog. ALBERTONI (*Arch. Ital. de Biol.*, vol. ix. pt. 1, p. 19, 1888).

Frogs when in act of coitus will submit to removal of cerebral hemispheres, &c., and to the most violent sensory stimuli, without abandoning their position, but relinquish it at once when the optic lobe is irritated.

CEREBELLUM.

17. Some Experiments in removing the Cerebellum. BORGHERINI (*Arch. Ital. de Biol.*, vol. ix. 1, p. 17, 1888).

Animals present oscillations of head and neck, and sometimes of trunk and limbs, with no loss of sensibility.

18. The Galvanic Vertigo. KUY (*Archiv f. Psychiatrie*, xviii. 3, p. 637, 1888).

Repetition and correction in some points of the experiments of Hitzig, Wundt, and others upon the sensations and eye-movements which result when electrodes are placed on the outer ear. Experiments also made upon a dog, from which Goltz had removed the whole vermis of the cerebellum. The animal exhibited the characteristic giddiness. Theoretical conclusion, that the seat of the giddiness is the cerebellum, not the semicircular canals.

SENSES.

19. The Influence of one Sense Stimulation on the other Sense Perceptions. URBANTSCHITSCH (*Pflüger's Arch.* xlii., 3 and 4, p. 154, 1888).

PSYCHOLOGY.

20. The Nervous System and the Mind. MERCIER (*London: Macmillan and Co.*, 1888, pp. 374).

A treatise on the laws of the healthy mind from a purely physiological or objective point of view, and intended as an introduction to the study of insanity. In the psychological sections, the mental states and phenomena are defined as adjustments of the organism to its environment, in terms easily understood by students whose previous training has been of a physiological rather than a metaphysical kind.

SPINAL CORD IRRITABILITY.

21. The Irritability of certain fibre-bundles in the Cord of New-born Animals. BECHTEREW (*Neurolog. Centralbl., March* 15, 1888, *No.* 6).

Advantage is taken of the fact that, in growing animals, the nerve-fibres are not irritable until they have acquired a myelin sheath. In the spinal cord of puppies the several bundles of fibres are stimulated as they become successively myelinated. Thus the cuneate fasciculus is irritable, while Goll's column still remains grey. It is irritable both proximally and distally, showing that the fibres of the posterior roots run down as well as up.

Two or three days after birth the fasciculus gracilis becomes irritable, reflex movements resulting from its stimulation, without indication of pain. The ground bundles of the anterior and lateral columns are irritable. In puppies the direct cerebellar tract is medullated at birth. Its central stimulation is followed by characteristic movements of the head and trunk. Centripetal fibres appear to be present also in the anterior part of the lateral column. The pyramidal tract becomes irritable in 11–13 days.

SPINAL GANGLIA.

22. Anatomy and Physiology of the Spinal Ganglia. GAD (*Med. Wochenschr., No.* 43, 1887).

Experiments similar to those made by Joseph (see these Abstracts 'BRAIN,' July 1887, p. 280). When the vagus is cut between the ganglion jugulare and the brain, the portion in the neck undergoes a partial degeneration which affects its centrifugal fibres, while the centripetal fibres connected with the ganglion remain intact. Cut in the neck, the peripheral stump becomes almost entirely atrophied and functionless in 4–6 weeks.

NERVES—CEREBRAL.

23. The Morphology of the Vagus Nerve. T. W. SHORE (*Journal Anat. and Phys.* xxii. pt. 3, *April* 1888).

NERVES—PERIPHERAL.

24. The Relation between the number of Fibres in the Nerve Trunks and the Body Weight in Mammals. WASZKIEWICZ (*Anat. Anz.* iii., *No.* 7, p. 206, *March* 1888).

25. Innervation of the Musculus Crico-Thyroideus. EXNER (*Pflüger's Archiv*, xliii. pt. 1, *March* 1888).

REFLEX ACTION—RESPIRATION.

26. Respiration Centre in the Medulla Oblongata, and the Conditions of its Action. LOEWY (*Pflüger's Archiv*, xlii., pts. 5 and 6, pp. 245–273, *Feb.* 1885).

REFLEX ACTION—THERMOTAXY.

27. The Influence of the Brain on Animal Heat and Fever. GIRARD (*Archives de Phys.* xx. 3, pp. 312–331, *April* 1888).

Concludes that while pricking a limited region of anterior part of brain induces rise of temperature, there is not a unique thermotaxic centre, nor can four or any other number of centres be localised with the exactness indicated by Ott (cf. 'BRAIN,' Jan. 1888, p. 550).

28. New Function of the Medulla Oblongata. Regulation of Temperature by Respiration. RICHET (*Archives de Physiologie*, xx. 2, p. 193, and 3 p. 292, *Feb. and April* 1888).

An investigation into the conditions determining and modifying the rapid respiration (polypnœa), by which an animal with impervious skin, *e.g.* the dog, increases the amount of water-vapour discharged from the lungs for the purpose of lowering its body-temperature. Polypnœa is not modified by section of both vagi, and is therefore not induced by intra-pulmonary excitation.

REFLEX ACTION—EYELID.

29. The Centre for Reflex Closure of the Eyelid. NICKELL (*Pflüger's Archiv*, xlii. pt. 11, *March* 1888).

An attempt to determine the anterior and posterior limits of this centre. Posterior limit found in the neighbourhood of the middle of the ala cinerea. Anterior limit coincides with that for the origin of the fibres of the facial nerve innervating the eyelid. Not considered to reach beyond the proximal border of the Pons Varolii.

REFLEX ACTION AND SENSORY NERVES.

30. Sensory Nerves and the Reflex-apparatus of the Spinal Cord. HÄLLSTEN (*Arch. f. Anat. u. Phys.; Phys. Abth.* 1888, pts. 1 and 2, p. 163).

A continuation of the series of observations already noticed (cf. these Abstracts 'BRAIN,' Jan. 1888, p. 541). The present Paper treats of the electro-tonic alterations in irritability in sensory nerves.

SYMPATHETIC—ANATOMY.

31. Topographical Study of the Cervical Sympathetic. DROBNIK (*Arch. f. Anat. u. Phys.; Anat. Abth.*, 1887, pt. 6, pp. 339–368, pl. xx., issued *Feb.* 1888).

A description with several illustrations of the topography and distribution of the sympathetic in the human neck, as determined by dissection.

SYMPATHETIC—CARDIAC.

32. The Centrifugal Nerves of the Heart. PAWLOW (*Arch. f. Anat. u. Phys.; Phys. Abth.* 1887, pt. 6, p. 498., pl. vii. and viii., issued *Feb.* 1888.)

SYMPATHETIC GANGLIA.

33. The Venous Tissue of Sympathetic Ganglia. RANVIER (*Comptes Rendus*, cvi., p. 574, *Feb.* 1888).

The arrangement of vessels in the ganglia is characteristic. Arteries are small and break up into a network of capillaries, the meshes of which are large enough to contain several cells. The veins are large, tortuous, varicose, plexiform, and end in culs-de-sac, into each of which several capillaries discharge. Like other nerves and ganglia, they have no lymphatic vessels, but a cavernous lymphatic system.

ELECTRIC-ORGAN.

34. Experiments on Curarized Torpedoes. GOTCH (*Proc. Physiol. Soc. Oxford, March* 17, 1888).

Direct stimulation of electric lobe is followed by discharge from electric organ in ·04 second. As noticed by Moreau, this is not affected by curare.

TERATOLOGY.

35. Histology of the Nervous Centre of a Diprosopus Tetrophthalmus belonging to the Human Race. MARTINOTTI and SPERINO (*Archives Ital. de Biol.* ix. 1, p. 8, 1888).

Of the four eyes the two median were contained in a single median orbit. Cranial cavity contained four cerebral hemispheres, one cerebellum, pons and spinal cord. On the anterior surface of the pons and medulla and the cord, as low as the dorsal region, was situated an anomalous median column containing segmentally arranged nerve cells, and considered by the authors as the vestige of a second atrophied spinal cord.

HISTOLOGY.

36. Anatomy and Histology of the Nervous Systems of Myzostoma. NANSEN (*Jenaische Zeitschr.* xxi. pts. 3 and 4, p. 267, *December* 1887).

37. The Structure and Combination of the Histological Elements of the Central Nervous System. FRIDTJOF NANSEN (Bergen, Jon Grieg, 1887, reprinted in English, from the *Bergens Museums Aarsberetning*, 1886).

Commences with an exhaustive account of the views held by histologists as to the minute constitution of nerve fibres and cells and their connection with one another. The author's investigations have been made on types of various classes of Invertebrates and Vertebrates, the nerve tissues of which he examined either fresh, in blood, isolated in acetic acid and glycerin, or dilute alcohol, or ammonium bichromate, or hardened and cut in sections. The sections were stained in osmic acid, carmine, hæmatoxylin, or coal-tar colours; or after Golgi's method, which the author is the first to apply successfully to the fish's spinal cord. Owing to the variety of methods used, the paper includes quite a treatise on the *technik* of nerve histology.

In all cases the nerve fibres (termed by the author "nerve tubes") are found to consist of an external consistent sheath, with viscous contents. The contents of this neuroglia sheath are divided up by a reticulate mesh-work of spongioplasm, the meshes of which form endless tubes containing the really nervous substance or hyaloplasm of viscous semifluid consistence. In the axis of the nerve the tubes of spongioplasm are often thicker walled and of smaller calibre than towards the periphery.

The "primitive tubes" traverse the protoplasm of the ganglion cells, giving them a concentrically striated appearance. The contents of each tube is more widely separated from that of its neighbours than in nerve fibres, fatty substance being apparently associated with the isolating spongioplasm. No direct anastomosis between the processes of ganglion cells. The bridges sometimes seen, are vestiges of cell division. Real unipolar cells exist, indeed they are almost the only ones found in certain invertebrates.

The "nerve tubes" and "primitive tubes" are the chief constituents of the dotted substance where they do not anastomose, but form only a more or less intricate web. The appearance of reticulation is due to the trans-section of the tubes. It is the author's opinion that "the irritation does not at all pass through any ganglion cells, but that after its arrival in the dotted substance, through subdivisions of the centripetal nerve tube, it is at once carried on into one or several centrifugal nerve tubes by the slender side-branches or fibrillæ joining them." The protoplasmic processes of the ganglion cells are not nervous. They are concerned with the nutritive functions of the cells.

HISTOLOGY—SPINAL CORD.

38. Microscopical Examination of Clarke's Column in Man, the Monkey and the Dog. MOTT (*Journal Anat. and Phys.* xxii. pt. 3, p. 479, *April* 1888).

Clarke's column is limited to those regions which Dr. Gaskell has shown give rise to the visceral-vascular system in the dog. Above, they are connected with the cerebellum. They must therefore be in connection below with afferent fibres. Author concludes that the direct cerebellar tract may constitute the connection between the viscera and the cerebellum. The distribution of the large multipolar cells of Clarke's column in man, as compared with the dog and monkey, suggests their connection with the maintenance of the erect posture.

HISTOLOGY—GANGLIA.

39. The Cells of the Spinal Ganglia in Mammals. DAAE (*Archiv f. Mikrosk. Anat.* xxxi. pt. 2, p. 223, pl. xiii. and xiv., *Jan.* 1888).

GANGLIA.

40. Physiological and Micro-chemical Observations with regard to the Cells of Peripheral Ganglia. ANNA KOTLAREWSKY (*Inaug. dis. Bern.* 1887).

HISTOLOGY.

41. The Histological Changes in the Peripheral Nerves, the Spinal Ganglia and Spinal Cord, which follow amputation. HOMÉN (*Neurolog. Centralbl.* vii. *No.* 3, *Feb.* 1, 1888).

Experimental study on thirty dogs. Times of incidence of the progressive atrophic changes recorded.

HISTOLOGY—NERVE-ENDINGS.

42. Nerve-end coils in the Frog's lung. SMIRNOW (*Anat. Anz.* Vol. iii. pt. 9, p. 258, *April* 1888).

HISTOGENY.

43. The Histogenesis of the Retina and Optic Nerve. FALCHE (*Archivio per le Sci. Med.* xii. 1, p. 1, 1888).

44. The Development of the First Nerve Paths in the Human Embryo. HIS (*Archiv f. Anat. u. Phys.; Anat. Abth.* 1887, pt. 6, p. 368, *issued Feb.* 1888).

As pointed out by His in former papers, the brain, spinal cord, and ganglia consist in the earliest period of their existence of isolated cells, without connection with one another or the periphery. Nerve fibres first grow out from the cells during the fourth week, some as processes of the cells of the spinal cord, others as processes from the cells of the spinal-ganglia. The latter cells are bipolar, sending one process into the spinal cord, the other towards the periphery. Before the nerve fibres are developed, a scaffolding of neuro- or myelo-spongium is formed for their support, also from processes of cells. The cerebral tube consists of two lateral thickenings, united dorsally by the "roof plate" and ventrally by the "floor-plate." The ventral half of the side wall His terms the "ground plate," the dorsal half the "wing plate." From the ground-plate cells grow out the fibres of the motor nerves. The sensory nerves originating in the ganglia grow into the spinal cord and brain. This latter proposition His is now able to maintain with great certainty.

METHOD.

45. Improvements in the Nitrate of Silver Re-action for the Nervous Centres. MARTINOTTI (*Archives Ital. de Biol.* ix., 1, p. 24, 1888).

With greater volume of nitrate of silver solution, longer immersion (15–30 days) at temp. of 25° C., and addition of 5 per cent. glycerine, it is possible to stain much larger sections than has been done heretofore.

EDITORIAL NOTE.—Students of Mental and Nervous diseases will be glad to know, that a Catalogue of old and new publications on these subjects may be obtained from Mr. Meurillon, bookseller, 16 Rue Serpente, Paris.

BRAIN.

OCTOBER, 1888.

Original Articles.

SPINAL LOCALISATIONS AS INDICATED BY SPINAL INJURIES.

BY WILLIAM THORBURN, M.D., B.S. (LOND.), F.R.C.S.,

Surgical Registrar to the Manchester Royal Infirmary.

IN 'BRAIN' for January, 1887, I published a series of nine cases of injury to the cervical region of the spinal cord, observed during the previous eighteen months in the Manchester Royal Infirmary, and was able to draw therefrom certain conclusions, which would appear to be of interest from the points of view both of physiology and of pathology. Since then I have had a further series of ten cases, which enable me to confirm and to extend the results then arrived at, and which I now bring forward as illustrating more especially the arrangement of the various nuclei situated in the brachial enlargement of the spinal cord. I have again to thank the Infirmary staff for permission to use these cases. They consist of seven fatal instances of fracture-dislocation, one of dislocation, which was cured by early reduction, and two of hæmorrhage into the spinal cord. My primary object in watching and recording them having been to ascertain more accurately than has hitherto been done the distribution of the paralysis resulting from injuries at different levels of the cord, I have arranged the first seven (fatal) cases in anatomical order, followed by the three others in which the seat of the lesion was inferential only, and have especially dwelt upon the exact distribution of the paralysis, but I have also referred to numerous other points of interest which they present. Further, this paper must be considered as a continuation of the one above spoken of, to which frequent reference will be made.

Going back then at the outset to the above-mentioned paper, I would recapitulate some of the results then arrived at. I presented the following cases:—1 (pp. 515 and 516). Two cases of fracture-dislocation between the fourth and fifth cervical vertebræ, in which all the muscles of the upper limbs, with the doubtful exception of the supra- and infra-spinati, were paralysed. 2. (p. 511). A case of fracture-dislocation between the fifth and sixth cervical vertebræ, in which all the muscles of the upper limbs were paralysed *except the deltoid, biceps, brachialis anticus, and supinator longus* and again doubtfully the supra- and infra-spinati, and in which the patient's arms consequently persistently maintained the position indicated by the photograph of which an engraving is here reproduced. And 3. Two cases (pp. 518 and 520) of fracture-dislocation between the fourth and fifth cervical and fifth and sixth cervical vertebræ respectively, in which, owing to the obliquity of the crush of the spinal cord, the fifth cervical nerve-root escaped on the right side only in the one case and on the left side only in the other, the result being that in the former the deltoid, biceps, brachialis anticus, and supinator longus escaped being paralysed on the right side, and in the latter they escaped on the left side only; the limb thus partially spared presenting in both instances a position

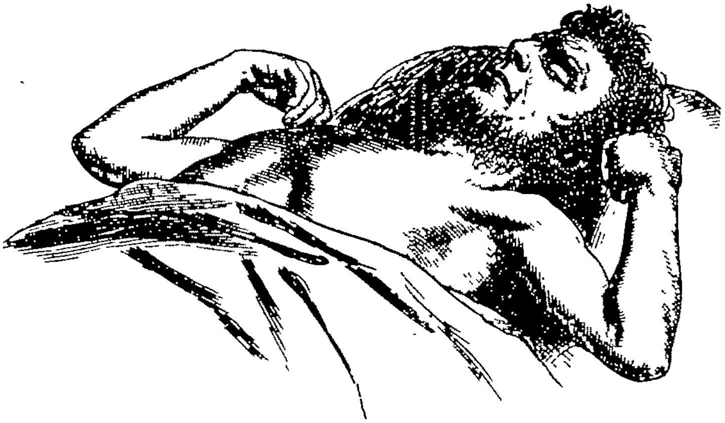

FIG. 1.—Position occupied by the limbs in a case of complete transverse destruction of the spinal cord immediately below the level of origin of the fifth cervical nerves.

similar to that of Fig. 1, whereas the opposite limb, whose fifth cervical root was involved in the lesion, was entirely flaccid.

Hence it was proved that, in injuries involving the cord below the fifth cervical nerve-root, we find the upper limb presenting a condition which is the exact antithesis of "Erb's paralysis" of the adult, or Duchenne's "obstetrical paralysis" as seen in the infant, and that consequently the limb occupies in such cases a position of abduction at the shoulder, with flexion of the elbow and supination of the wrist. It was further concluded, for reasons to which it is unnecessary again to refer, that the fourth cervical nerve innervates the supra- and infra-spinati muscles. These conclusions the present paper will be found to confirm, but we shall now also be able to trace the arrangement in the brachial enlargement of a number of the other spinal centres for the various muscles of the upper limb. In endeavouring to work out this matter I have entirely ignored the results as to localisation previously arrived at by other methods, and have confined myself entirely to the evidence yielded by my own cases, the more so as the conclusions are consistent with the majority of previous observations.

Before proceeding to give the evidence, I shall now tabulate provisionally what I am so far led to believe to be the arrangement, from above downwards in the cord, of the muscle nuclei, as follows:—

Supra-spinatus and infra-spinatus Teres minor (?)	4th cervical nerve.
Biceps Brachialis anticus[1] Deltoid Supinator longus Supinator brevis (?)	5th cervical nerve.
Subscapularis Pronators Teres major Latissimus dorsi Pectoralis major Triceps Serratus magnus	6th cervical nerve.
Extensors of the wrist	7th cervical nerve.
Flexors of the wrist	8th cervical nerve.
Interossei Other intrinsic muscles of the hand	1st dorsal nerve.

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[1] I have not identified the brachialis anticus, but from Erb's researches and others, assume it to be in close connection with the biceps

The various cases which follow will, I believe, substantiate the above arrangement.

CASE 1.—A man aged 35 was admitted under Mr. Heath's care on June 18, 1887. He had the night before admission fallen down about ten steps on to his head, as evidenced by bruises on the left side of the cranium. As the man died in a few hours, I was obliged to obtain my information from Mr. Thompson, the house surgeon.

At the back of the neck was a large amount of effused blood, but the spines of the vertebræ were distinctly felt, that of the sixth cervical being apparently displaced slightly to the right. The head itself was not twisted, and could be turned from side to side, nor did the neck appear abnormally flexed or extended. There was only slight pain in the back of the neck.

The lower limbs and trunk were completely paralysed; the arms were held rigid with the elbows abducted, and the hands lying on the epigastrium extended and prone. Anæsthesia was complete below a point about two inches above the nipples, being also complete over the shoulders and in the upper limbs. The pupils were reduced to fine points, but the size of the palpebral fissures was not noted. Reflexes were not observed. The urine was retained, and the penis turgid.

As the man was being placed in bed respiration became slow and gasping, with distinct action of the scaleni and sternomastoids; the patient grew cyanotic and respiration then stopped, the pulse ceasing about half a minute later. There was no *post-mortem* examination.

The above case does not carry us very far, but it is clear from the facts adduced, and especially from the anæsthesia affecting the whole surface of the upper limbs, that almost the entire brachial region was affected. With the exception of the abduction of the shoulders and flexion of the elbows, the arms were in the position of rest, resulting from complete paralysis. Hence then the biceps and deltoid had escaped, but the supinators and all muscles mentioned below them in our table were paralysed; that is to say, the hæmorrhage resulting from the crush of the cord had extended partially into the territory of the fifth root, destroying the centre, at its lower part, for the supinator, but sparing the biceps and deltoid above.

CASE 2.—A. B., aged 23, was admitted to the Manchester Infirmary under the care of Mr. Jones on April 7, 1888. He was playing football, and, while stooping forwards with his head thrown up in order to "tackle" an opponent, came into violent collision, his head being jerked backwards. He was immediately paralysed.

On admission the spine of the seventh cervical vertebra was felt to be distinctly depressed, but with little or no lateral displacement. There was a good deal of swelling about the back of the neck, and the patient complained of great pain. The lower limbs and trunk were completely paralysed, respiration being diaphragmatic. In the upper limbs the only muscles not paralysed were the flexors of the elbow. They at first lay extended and close to the body, but the patient bent the elbows so as to bring the hand up to the shoulder when told to try to move them. The pupils were slightly contracted.

An attempt was at once made to reduce the dislocation by extension, followed by flexion of the neck, and a distinct jerking was felt by the assistants who were steadying the neck. The patient expressed himself as feeling his pain relieved, but no other change was produced. Later on another attempt at reduction was made with chloroform, but with equally little success. The patient was now put to bed, the head being steadied by pillows and sandbags. At this time the temperature was below 95° and the skin felt cold; the pulse was strong and regular; the penis was turgid, the urine retained.

About 4 A.M. on the following day, the temperature began to rise rapidly; the skin was hot, flushed, and perspiring; and the patient became delirious. At 6 A.M. the temperature was 106°·6, at 8 A.M. 108°, and at 9.30 110°, at which time he died comatose.

There was no *post-mortem* examination. For notes of the above I am indebted to Mr. Stocks, the house surgeon.

This case is also somewhat unsatisfactory, as the accident happened on Saturday night, and the man died on the Sunday morning, so that it was not very fully observed. It indicates sufficiently the high position of the nucleus for the flexors of the elbow, and shows that the deltoid and supinators, also supplied by the fifth root, were paralysed. Thus, then, we are led to place the deltoid nucleus below that of the flexors of the elbow, a conclusion confirmed by a case mentioned in my previous paper, where at first the entire fifth root was spared, but where, as myelitis extended upwards, "the contraction of the deltoid muscles, which had maintained the abducted position of the arms, ceased, and the elbows fell to the sides, the elbow-joints themselves remaining flexed."[1] These two cases, then, compared with the cases referred to in the previous paper, carry us so far as to assign the order—

$\left\{\begin{array}{l}\text{Biceps.}\\\text{Brachialis anticus.}\end{array}\right.$
Deltoid.
Supinator longus.

CASE 3.—J. C., a labourer, aged 38, was admitted to the Manchester Royal Infirmary under the care of Mr. Hardie on October 25, 1887. On the afternoon of this day he had fallen from a waggon for a distance of about six feet, striking the back of his shoulders and head. He was immediately paralysed, but did not lose consciousness.

I did not see him until the following day, but am informed by the house surgeon that his condition on admission was the same as when he came under observation.

On Oct. 26 he had pain across the shoulders, shooting down the arms to the elbows, but no tenderness of the spine or pain on pressing down the head. There was some arching backwards of the cervical vertebræ, but no lateral deviation and no obvious deformity of the spinous processes.

The lower limbs were absolutely paralysed, as were the abdomen and thorax, respiration being diaphragmatic, jerky, 18 per minute, with subjective sense of dyspnœa and cough. The left upper limb was abducted at the shoulder, with the elbow flexed and the hand across the chest, the right being completely flaccid. On the left side he had the power of voluntary contraction of the biceps, supinator longus, and deltoid, but not of the other limb muscles, whereas on the right side all the muscles were paralysed. On both sides the pectorals and latissimi were paralysed, but on the left side the supra-spinatus and infra-spinatus could be made to contract, whereas on the right they could not. Hence of the intrinsic and extrinsic muscles of the upper limb all were paralysed on the right side, but the spinati, deltoid, biceps, and supinator longus had escaped on the left, giving the limb the above-described characteristic position. The neck muscles were normal on both sides. Both palpebral fissures and pupils were smaller than usual, but not so markedly as in many cases.

Anæsthesia extended as high as the third rib in front and the sixth cervical spine behind, but on the right side he retained sensation over the region of the deltoid and slightly beyond it, and on the left side it extended over the area of the deltoid and thence down the outer side of the limb to about the root of the thumb, becoming, however, very vague at the lower part. The anæsthetic boundary was not very sharply marked, and he had no hyperæsthesia.

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[1] As the above is passing through the press, I have seen another case in which ascending traumatic myelitis caused paralysis of the deltoid *before* that of the biceps, a condition, I admit, at first considered improbable

The knee-jerk and plantar, cremasteric, gluteal, and epigastric reflexes, were absent. The skin felt dry and warm, although the temperature was normal. Urine was retained. The penis was turgid, but less so than on the previous day. Pulse 66, feeble, and with a very marked respiratory wave, being full and soft towards the end of inspiration and very small towards the end of expiration.

The man's condition being clearly otherwise hopeless, Mr. Hardie determined, after consultation with several of his colleagues, to "trephine" the spine, which was done at 4.20 P.M. on the afternoon of this day, about twenty-four hours after the injury. For the subsequent notes, I am indebted to Mr. Bannister. Chloroform having been administered, a vertical incision about four inches long was made over the cervical spines, having its centre opposite to the fifth. The muscles being cleared from the laminæ, an interval of about a quarter of an inch was found between the fifth and sixth spines, and the fifth vertebra appeared to be slightly displaced forwards. The laminæ of the fifth and sixth were now removed by bone forceps, when the dura mater was exposed, presenting a perfectly normal appearance without any trace of hæmorrhage. As nothing further could be done, the dura was not opened, but the muscles were brought together by deep catgut sutures, a large drainage tube inserted, and the wound closed over a smaller superficial drain. The carbolic spray was used throughout, and wood wool pads used as a dressing. After the operation brandy was given at frequent intervals.

At 6 P.M. the pulse was 54 and feeble; temperature 96°; respirations 13, and still purely abdominal. At 10 P.M. pulse 74; temperature 98°·6, respirations 16. The patient passed a fairly good night, but in the morning the temperature was 103°·4. About 3.20 P.M. on the afternoon of the 27th he became suddenly much worse; the respirations became very feeble and then stopped, the abdomen being uniformly distended; the heart-beats, which were very feeble and infrequent, continued for about ten minutes after respiration had ceased, but stopped at 3.35. At 3.40 the temperature in the mouth was 104°, and the body, which had previously felt very hot, was already noticed to be beginning to cool.

The *post-mortem* examination was made by Dr. Harris. The disc between the fifth and sixth cervical vertebræ was found to be ruptured, the former bone projecting very slightly forwards. No fracture was discovered. The dura mater was uninjured, but the cord was flattened opposite the seat of injury, and was much contused for about an inch above and below, containing hæmorrhages in its substance and in the central canal; elsewhere its structure was normal. The first and second bones of the sternum were also partially separated, and the lungs much congested.

This is another case similar to those already mentioned, in which the cord was crushed just above the sixth cervical root, but on the right side the fifth root was also affected. Unfortunately, I was unable to be present at the *post-mortem*, and the exact pathological cause of this obliquity was not noted, but doubtless the hæmorrhage which occurred in the substance of the cord extended somewhat higher on the right side. The case merely confirms our previous conclusions.

CASE 4.—J. E. C., aged 33, was admitted into Mr. Heath's wards on March 25, 1887, at 2.30 P.M. He had shortly before been "larking" with some friends, and had another man seated upon his shoulders, when he was pushed backwards in the face against a high counter. The result was that his neck was twisted backwards, the man falling from his shoulders. For the first few minutes he only noticed pain in the back of the neck, but then his legs began to feel weak and he lay down: within ten minutes the lower limbs were completely paralysed and insensitive, and he found that he could not straighten the left fore-arm.

When admitted, there was paralysis of the lower limbs, and the left upper limb was only partially movable, being flexed at the elbow and somewhat abducted at the shoulder. The right limb appeared to retain more power. The temperature at 4 P.M. was 94°·2, at 8 P.M. 97°·6, at midnight 99°·2, near which point it remained for some days. He was placed on a water bed and the urine withdrawn by the catheter.

On the following morning he complained of pain in the back of the neck, but there was no local deformity, and no pain on pressing down the head. The lower limbs were absolutely paralysed, as were the abdominal and thoracic muscles, respiration being diaphragmatic.

The left upper limb lay with the fingers flexed, the wrist straight, hand prone, elbow flexed, and the humerus abducted to an angle of about 45° with the body: he often raised the humerus above the shoulder, otherwise retaining the same positions, but with the wrist becoming semi-prone. He could not extend the elbow nor flex nor extend the fingers, but on attempting to flex the fingers there was slight extension of the wrist; he had also slight power of flexion and extension of the wrist, but this movement was extremely limited: pronation and supination were fairly good: the pectoralis major, latissimus dorsi, and apparently the subscapularis contracted very feebly; the deltoid, biceps, and supinator longus were but little if at all impaired.

On the right side the condition was practically the same, except that there was rather more power in the wrist. On taking a deep breath, the sterno-mastoids, trapezii, and levatores angulorum scapularum contracted very distinctly.

As regards sensation, there was "a feeling of heat" in the left palm and fore-arm. Anæsthesia was nowhere absolute, but was almost so below the third ribs, and in the upper limbs internally to a line running down the centre of the arms and fore-arms to the styloid processes of the ulna, both before and behind. Below these limits there was absolute analgesia, but a vague sensation was conveyed by tickling. The limit was very ill-defined, and above it was an indistinct hyperæsthetic zone.

There were no superficial reflexes nor tendon reactions.

Urine was retained, and had to be drawn off by the catheter: its sp. gr. was 1024; it was neutral, and contained neither sugar, albumin, nor any deposit. The bowels had not been moved since admission. The respirations were at the rate of 19 per min., with no sense of dyspnœa, but slight cyanosis. Pulse 69, full and soft. Skin dry and warm. The penis was turgid. Both palpebral fissures and pupils were small, and there was no dilatation of the pupils on pinching the neck.

Two days later, the paralysis of the upper limbs was of equal extent on both sides. There was complete loss of power in all the intrinsic and extrinsic muscles except the deltoid, biceps, supinator longus, and subscapularis, the elbows being flexed, the humeri abducted but not rotated outwards, and the hands thus lying across the chest. The pectoralis major, latissimus dorsi, and teres major had ceased to act at all. The region of anæsthesia in the upper limbs had also extended. The temperature was normal. The optic discs were examined, but presented no abnormality. Priapism was less marked.

On the 4th day the urine contained a little pus (and therefore albumin), but no sugar. Its reaction was acid.

On the 5th day the optic discs appeared less well defined than before, and presented some venous congestion.

On the 6th day the subscapularis and supinator longus could no longer be felt to contract, but the arms usually occupied the same position as before, although when in sleep they were often raised above the shoulder. He complained much of a sense of pain and stiffness in the upper limbs. The optic discs were more vascular and were hazy—an observation which was confirmed by Dr. Little, who held, however, that there was no neuritis.

For a few days sensation in the trunk and lower limbs now seemed to improve slightly, but the patient became weaker, and was frequently somewhat delirious at night. The temperature ranged from 100° to 101°.

On the 11th day were noted tremors of the paralysed muscles of the upper limbs. A day later, the biceps and deltoid of the left side were found to be beginning to fail, and the humerus was less abducted than formerly. At the same time sensation began to fail again.

On the 14th day the left upper limb was absolutely paralysed and anæsthetic, the humerus lying by the side, the elbow at a right angle. On the right side, the only muscle whose contraction could be felt was the deltoid. Ecchymoses had formed on the heels, toes, and malleoli.

On the 15th day we found distinct knee-jerk on the right side, and on the left a slight reaction could be obtained.

A few days later he developed symptoms of pneumonia, and the condition of the urine became much worse. The temperature, which had regained the normal on the 15th day, again began to rise on the 23rd, keeping a fairly steady upward curve until April 23rd or the 29th day, when it reached 104°·6 and when he died. During the latter period there appeared to be more wasting in the fore-arms and hands than in other parts of the body. On several occasions also it was noted that the passage of the catheter was accompanied by reflex contractions of the sartorii.

At the *post-mortem* examination I found some separation of the fifth and sixth cervical spinous processes. The body of the fifth was partially dislocated forward, forming a very obtuse angle with that of the sixth, and overlapping the latter by about a line anteriorly. The articular processes were ***in situ***. The upper and anterior margin of the body of the sixth vertebra was very slightly ground off, but there was no other fracture. The vertebral canal contained a little dark clotted blood at the seat of the lesion. The cord was here partially compressed and very pale, all distinction of white and grey matter being lost. This condition extended only for about an inch, and elsewhere the cord presented no macroscopic abnormality. No nerve-roots were crushed. There were marked cystitis and double pneumonia.

We have here another case in which the cord was crushed below the fifth cervical nerve-roots, the muscles supplied by which retained their power for a considerable time. The case differs, however, from those previously mentioned in that the annihilation of the functions of the cord below the lesion was less complete, as evidenced by the fact that anæsthesia was not absolute, and that some power remained in muscles supplied by nerve-roots distinctly below the lesion. The examination on the second day showed that, whereas the deltoid, flexors of the elbow, and supinators retained a very fair amount of power, the other muscles of the limb were paralysed to a varying extent. Thus next in order of power we find the subscapularis, pectoralis major, latissimus dorsi, and pronators, between which no distinction could be drawn, and we find also that the extensors of the wrist still retained a little power. On the fourth day we have paralysis of all the muscles of the plexus, except the "fifth-root group" and the subscapularis, the latter preventing the outward rotation of the humerus seen in Fig. 1. Again, on the sixth day the subscapularis and supinator fail, and later the deltoid and biceps begin to do so. As there can be little doubt that myelitis was extending upwards, we may then venture to give the following as the order in which the muscles here lost their power:—

1. Intrinsic muscles of the hand and flexors of the wrist immediately completely paralysed.
2. Extensors of wrist almost absolutely paralysed on second day.
3. Pectoralis major, latissimus dorsi, teres major and pronators following before fourth day.
4. Subscapularis retaining power longer than any muscles but those of the fifth root.

This, it will be found, is in accordance with our scheme of localisation. The position of the pronators is the most doubtful and the most difficult to ascertain, as the action of gravity interferes with the attitude of the hand.

CASE 5.—E. M., a woman aged 43, was admitted under the care of Mr. Heath on December 5, 1886, on which day about 11 A.M., while intoxicated, she had fallen down some twelve steps. She was unconscious for a short time, and on recovering found that she had lost all power of moving the trunk and limbs. She was brought to the Infirmary, and admitted about 5.15 P.M. on the day of the accident.

On admission she was very drunk, and a complete examination was impossible. The skin felt very cold and the temperature was below 95°, but no thermometer of a lower register was at hand. The pulse could not be felt. There was no sweating. She complained of pain behind the shoulders, but there was no deformity. The lower extremities and the left upper limb were entirely paralysed and flaccid. On the right side the shoulder was abducted, and rotated somewhat outwards, the elbow being flexed, the wrist and fingers flaccid. Respiration was diaphragmatic. The urine was retained. Owing to the vagueness of her answers, it was impossible to ascertain the limits of sensation, but there appeared to be anæsthesia below the neck. The right pupil was small, distorted, and immovable, owing to posterior synechia; the left was small, but responded both to light and accommodation.

When I examined her on the following morning, the patient presented the following symptoms:—The intellect was perfectly clear, and she gave rationally the above account of her accident. She presented total paralysis of all four limbs, and of the muscles of the chest and abdomen. She had no power of moving even the shoulders; the pectoralis major, deltoid, supra- and infra-spinati, latissimus dorsi and subscapularis, as well as the intrinsic muscles of the upper limb, being quite flaccid on both sides. There was no tendency to rotation of the humerus, either inwards or outwards. On the other hand, the trapezii and sterno-mastoids were tonically contracted.

Respiration was diaphragmatic, but there was no indication of dyspnœa and the voice was not weak. The urine had to be drawn off by means of the catheter, and the bowels had not been moved since admission. The palpebral fissures were smaller than usual, giving the patient a sleepy look. The right pupil was as described above, but presented some further contraction on exposure to light.

She complained of a sense of coldness in the left foot, and of great pain in the back of the neck and across the shoulders, pain being aggravated by any movement of the shoulder-joint. The limbs and trunk were completely anæsthetic, there being a band of hyperæsthesia about 1½ in. wide, extending in the form of a collar across the chest, at the level of the second rib and outwards across the deltoids. Above this line sensation was normal. It was not possible accurately to define the limit of sensibility behind, owing to the necessary movement causing great pain in the neck. The firmly-contracted trapezius was painful on deep pressure. No pain was caused by very firm downward pressure on the head.

The plantar reflex was exaggerated, the slightest tickling of the sole causing clonic spasms of the limb. No abdominal cutaneous reflex could be obtained. There was no knee-jerk, nor ankle clonus.

The cranial nerves presented no abnormality. The right optic disc was normal; the left could not be seen, owing to a commencing cataract.

The pulse was very irregular and intermittent, full, soft, and beating at the rate of 108 per minute. The heart-sounds presented nothing abnormal.

Respirations were 28, and, as above stated, entirely diaphragmatic, the chest falling in on inspiration.

The temperature at 8 A.M. was 95°. When examined at 12 midday, it was 97°, but the skin of the trunk and lower limbs felt warm and somewhat dry.

The urine had a sp. gr. of 1024, was acid, contained no albumin and no deposit. It decolorised but did not precipitate Fehling's solution. The nurse informed me that she had a considerable amount of vaginal discharge.

The temperature now began to rise steadily, and at 9 P.M. on the same day reached 101°·6, at which it remained until death, which occurred at about 8 A.M. on the following day; that is, about forty hours after the accident. Death resulted from failure of respiration.

At the *post-mortem* examination we found a separation between the laminæ of the sixth and seventh cervical vertebræ, the whole of the ligaments uniting the arches being ruptured, and the sixth vertebra being carried forwards and to the right.

On removing these bones, the left inferior articular process of the sixth was found completely slipped off the superior articular process of the seventh, whereas on the right side it was only displaced forwards to a slight extent. There was an obliquely horizontal fracture across the body of the seventh vertebra, extending from the cartilage above it forwards and slightly downwards to a line about one-fourth of the distance from its upper to its lower border in front. The piece of bone thus separated was, with its adjacent cartilage and the sixth vertebra, carried forwards and somewhat downwards and to the left. On opening the spinal canal there was found extravasated and coagulated blood external to the dura mater, throughout the entire distance from the sixth cervical vertebra to the inferior termination of the canal. The disc between the sixth and seventh vertebræ had been crushed and forced backwards into the spinal canal, forming a shelf about one line in depth, which pressed upon the cord, but not to any great extent. The roots of the seventh nerves were crushed. The cord was slightly pressed upon by the above-mentioned projecting disc, but was nowhere compressed by the extra-meningeal hæmorrhage. Opposite the body of the sixth vertebra it presented a slight swelling of a bluish colour. Its substance was here quite diffluent and full of dark blood. From this point upwards as high as the origin of the third cervical nerves the cord was very soft and contained small extravasations of blood, mainly in the grey matter and especially in the central canal, these appearances being less marked at the upper than at the lower part. Below the sixth intervertebral disc the cord was slightly softened for the distance of about an inch. Elsewhere it presented no abnormality to the naked eye. The membranes appeared normal.

In this case no information of value is to be obtained as to the question of localisation, as the hæmorrhage into the cord extended so rapidly as almost at once to paralyse the cord throughout the entire brachial region, although the lesion of the vertebræ was at the level of origin of the seventh cervical nerves. For a short time after admission the fifth cervical nerve on the right side escaped, but this also was destroyed before I saw this patient, and death soon followed. The extensive central hæmorrhage found at the *post-mortem* amply explains this condition, and the case is chiefly of use as showing that the damage to the cord may almost at once extend to a considerable distance above the vertebral lesion.

CASE 6.—W. T., aged 28, a labourer, was admitted to the Manchester Royal Infirmary under the care of Mr. Wright on October 3, 1887. Two days previously he had been at work on the roof of a house when he fell, a distance of about eleven feet, on to a block of stone, which struck him just between the shoulder blades, causing his head to be jerked backwards. He felt very sick, but did not vomit, and then found that he was powerless to move either his arms or his egs. During the two days which he remained at home no change can be ascertained to have occurred in his condition.

On admission he presented complete paralysis of the lower limbs and of the abdominal and chest muscles, respiration being diaphragmatic. There was pain over the first dorsal vertebra, but not elsewhere, and no pain on pressing down the head. The upper limbs lay across the chest with the elbows close to the sides, and flexed, as in the annexed sketch made by the house surgeon, Mr. Bannister. On both sides the flexors of the wrist and fingers were completely paralysed, and he had no power of grasping; on the right side the extensors were also paralysed, but on the left they retained very slight power. The triceps, pectoral muscles, and serratus magnus could be felt to contract, but only very feebly so. The deltoid, biceps, and supinator longus were more powerful than any of the other muscles of the upper limb, but were also weak.

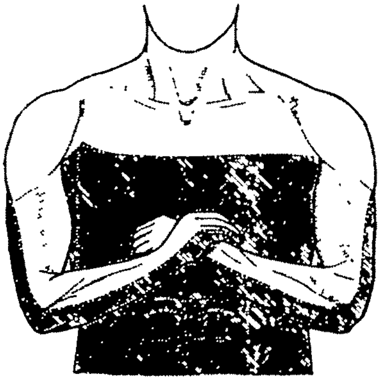

FIG. 2.—Position occupied by the limbs in a case of complete transverse destruction of the spinal cord immediately below the nuclei for the subscapularis muscles.

Both palpebral fissures and pupils were small, the right being more markedly so.

The bladder was much distended, and on passing a catheter a large amount of normal urine was drawn off.

Anæsthesia was very clearly defined, extending over the lower limbs and up the trunk as high as the level of the second rib in front, and to the second dorsal spine and spines of the scapulæ behind. In the upper limbs it affected the little finger and inner part of the ring finger, the ulnar side of the hand and fore-arm, and the intero-posterior aspect of the arm, as shown in the sketch by the shaded portions. The rest of the upper limbs was sensitive. About the boundary of the anæsthetic region was slight hyper-æsthesia, more marked in some parts than in others.

The penis was semi-erect. Temperature 99°·8.

On the following day the urine was ammoniacal and of a deep purple colour from admixture of blood. This profuse hæmorrhage from the kidneys continued for some days, during which time the patient became gradually weaker, but it ceased on October 10; the urine continuing, however, to be ammoniacal and containing some pus.

On October 9 the temperature in the right auditory meatus was found to be 101°·2 as against 100°·2 on the left side, this observation coinciding with the more marked diminution of the pupil and palpebral fissure on that side due to paralysis of the sympathetic.

On October 20 the optic discs were examined and found to be normal.

On October 25 the right upper limb lay across the chest with the elbow to the side, the hand on the upper part of the sternum and the fingers closed. When asked to move it, he only rotated the humerus outwards, but as he did so the supra-spinatus and infra-spinatus could be felt to contract. He still had very feeble power of flexing the elbow, but neither the biceps nor the supinator longus could be felt to contract, and he had lost all power over the deltoid. All the other muscles of the limbs were absolutely paralysed. On the left side the elbow was also close to the chest, but less flexed, so that the fore-arm was at about a right angle to the upper arm. On this side he had more power of flexing the elbow, and could abduct and adduct as well as rotate the shoulder, there being obvious contraction of the deltoid, biceps, and spinati muscles; he had also some feeble power of pronation. On breathing deeply, the levatores angulorum scapularum could be felt to contract. Anæsthesia was the same as on admission.

On November 1 he was much weaker, and it was found almost impossible to draw off the urine, owing to the catheter continually becoming blocked with stringy mucus. Severe vomiting and hiccough also troubled him greatly, and he refused all food. The temperature throughout the case varied from about 99° to 101°. On November 3 he died exhausted, having survived the accident thirty-one days.

At the *post-mortem* examination, which was made by Dr. Harris, there was found rupture of the disc between the last cervical and first dorsal vertebræ, the parts being very movable, but no fracture was detected, and when the *post mortem* was made there was no displacement. The cord opposite the seat of injury was almost diffluent, and softening extended throughout almost the whole cervical region and for some short distance below, but the remainder of the dorsal region showed no abnormality. The bladder was much enlarged and its walls thickened: on its mucous membrane was a thick, gritty, yellowish-white membrane which came away as a distinct cast: the peritoneal surface was inflamed and adherent to the sigmoid flexure and omentum. In the kidneys were the appearances of acute parenchymatous nephritis.

Here as in Case 5 we find the cord lesion extending above the site of dislocation before the patient came under observation. As before we have the deltoid, biceps, and supinator fairly powerful. The triceps, pectoral muscles and serratus magnus also retain a fair amount of power and appear to have received about an equal amount of damage, justifying their being placed near together in our table. It will be noticed that the attitude shown in Fig. 2 differs from that of Fig. 1 owing to the adductors (chiefly the pectoralis and subscapularis) of the humerus not being completely paralysed. At the end of three weeks we find on the left side that the supra-spinatus, infra-spinatus, deltoid, biceps, and apparently the supinators, subscapularis, and pronators, retain some power; whereas on the right side the supra-spinatus and infra-spinatus are the most powerful muscles, the flexors of the elbow are not quite paralysed, but all the other muscles are. Again therefore the arrangement confirms that of our table, as far as it goes.

Case 7.—J. S. was admitted to the Manchester Royal Infirmary under the care of Dr. Morgan on February 5, 1887, about 10.30 P.M. He had been at a place of entertainment, and when drunk had fallen from a gallery some ten feet in height, but it was not known how he struck himself.

It was nearly twenty-four hours after his admission when I first saw him, and when he presented the following symptoms. The lower limbs and trunk were completely paralysed, respiration being diaphragmatic. In the upper limbs movements were normal, with the following exceptions:—The intrinsic muscles of the hand were paralysed, the fingers being flexed and their extensors (*i.e.* the interossei and extensor communis digitorum) paralysed, whilst any attempt to completely flex them caused slight extension of the wrist; the wrist was held straight, both its extensors and flexors, but especially the former, being weak. On the right side the extensors of the wrist were weaker than on the left. The lower limbs were completely anæsthetic, as was the trunk, as high as the level of the second rib in front and the spine of the seventh cervical vertebra behind. In the upper limbs anæsthesia was limited with the greatest accuracy to the inner side of the arm and fore-arm, being bounded in front by a line running vertically from the front of the deltoid opposite the second rib, down the centre of the biceps and fore-arm to the middle of the wrist; similarly behind the boundary was a vertical line down the centre of the triceps and back of the fore-arm to the centre of the wrist. In the hand the limiting line sloped, both in front and behind, from the centre of the wrist to the cleft between the fourth and fifth fingers, the little finger being completely anæsthetic, the others not at all so. There was no trace of hyperæsthesia.

Superficial and tendon reflexes were all absent. Both palpebral fissures were distinctly small, as were the pupils, the latter being as usual especially notable in a dim light. The pupils moreover did not dilate on pinching the skin, but they showed very marked dilatation on each inspiration. Urine was retained. Penis turgid. Pulse 80. Temperature, *mane* 98°, *vesp.* 99°·4.

On the following morning some additional points were noted. He had no pain anywhere, but a sense of stiffness in the upper limbs, and no pain was caused by pressing or jerking the head downwards. The seventh cervical spine was thought to be rather more prominent than usual. Respiration was mainly diaphragmatic, aided by the upper scapular and sterno-mastoid muscles; it was at the rate of 24 per minute, without sense of dyspnœa. He had slight cough and some collection of mucus in the bronchial tubes. The plantar, cremasteric, abdominal, and epigastric reflexes, and the tendon reactions at the ankle, knee, wrist, and elbow, were all absent. The urine had a specific gravity of 1028, and contained neither sugar nor albumin. The pulse was 80, soft, and feeble; sphygmographic tracings taken from the radial and the posterior tibial arteries showed nothing unusual in either vessel. The heart was normal. The skin felt dry and warm, although at this time the axillary temperature was only 96°·2, but the chest and abdomen were moister than the limbs; it presented no redness or trace of soreness.

On the third day he felt himself better, especially as he had been troubled with vomiting, which had now ceased. He had less power over the fingers and wrist, the former being flexed, the latter extended and without power of movement. Dr. J. C. Thresh, the clinical clerk, called my attention to the fact, that the urine (specific gravity 1031) completely solidified with cold nitric acid, owing to the formation of nitrate of urea. The optic discs were a little hazy, with slight venous congestion.

On February 10 the discs were again examined by Dr. Little, who noted these symptoms, but could not definitely assert that there was any pathological change.

These symptoms continued without any important change until February 11, when it was found that on both sides the patient had lost the power of rotating the wrist or extending the elbow, and that no contraction could be felt in the triceps muscles. The fingers and wrists were flexed and completely paralysed. The pectoralis major on either side was also now paralysed, but the latissimus dorsi, teres major, and subscapularis could be felt to contract on voluntary effort. The position of rest of the upper limbs was now as follows: the humerus abducted almost to a right angle and slightly rotated inwards, the elbows fully flexed, the wrists prone, and the hands across the chest with the wrists and fingers flexed. Breathing was much more difficult: he presented some signs of mental hebetude: the whole body was dry and cold, and the urine was still loaded with urea and phosphates (in solution), having a specific gravity of 1033.

On February 12 the subscapularis could no longer be felt to contract and the latissimi were weaker, the teres major remaining about the same as the day before. The humerus was now rotated *externally* more readily than *internally*, thus differing from the condition of the previous day, and it was a greater effort to bring it towards the side. A little redness had appeared over the malleoli.

On February 14 we found that the boundary of absolute anæsthesia was the same as on admission, but some dulness of sensation extended further outwards on the limb. The motor symptoms were unaltered. The optic discs had become more indistinct than before, and the small vessels were quite obscured. Dr. Little concluded that there was now definite optic neuritis.

During the evening of this day he vomited freely both food and "coffee grounds," and, becoming delirious, died about 1 A.M. on February 15, the tenth day after the accident.

The annexed chart (fig. 3, p. 308) shows the very low temperature which was maintained during the entire duration of the case.

At the *post-mortem* examination we found a fracture of the anterior and upper part of the body of the first dorsal vertebra, a small portion being clipped off and remaining attached to the disc above. The seventh cervical vertebra was displaced forwards, carrying with it the above-mentioned fragment of the first dorsal.

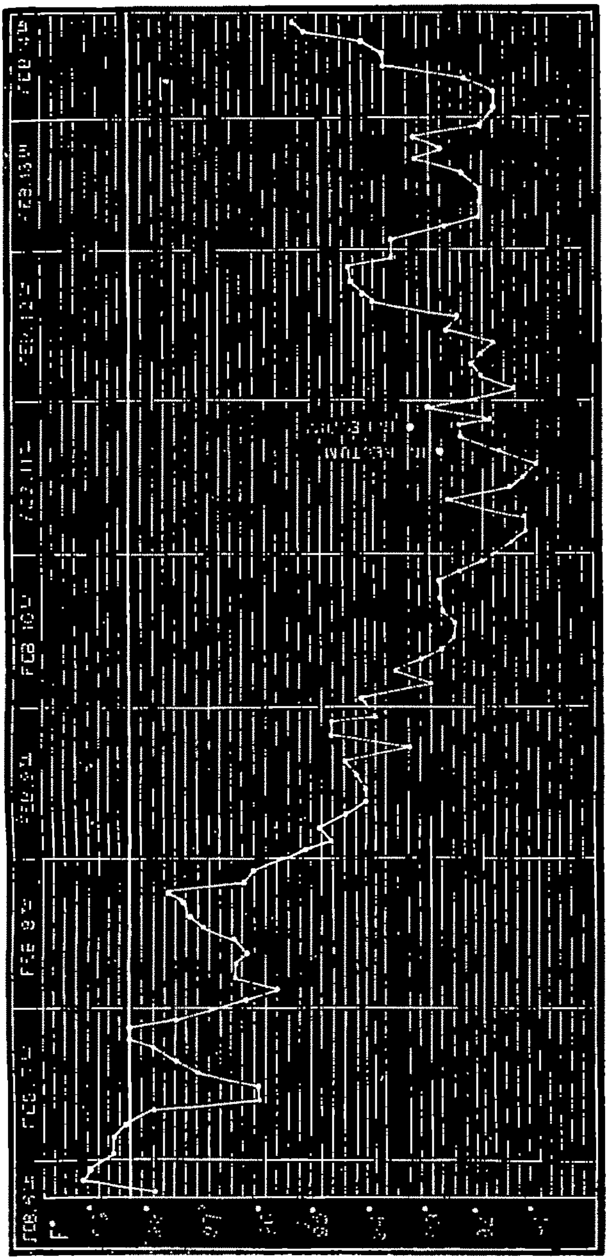

FIG. 3.—Temperature-Chart in a case of Fracture-dislocation of the 7th cervical vertebra. The temperatures were registered every two hours. At 12 2, 4, &c., till Feb. 9th; at 1, 3, 5, &c. till the 13th at noon; then every three hours. The thermometer was carefully tested and found to be practically correct.

The laminæ of these two vertebræ were separated behind, the articular processes of the first dorsal being posterior instead of anterior to their fellows of the seventh cervical. On opening the spinal canal there was found slight effusion of blood between the bone and the dura mater. The cord was compressed at the level of the first dorsal vertebra, and softened for a short distance above and below the site of compression, its centre being occupied by an effusion of blood reaching as high as the fifth cervical nerve-roots, in the form of a narrow cone. There was some suppuration between the anterior common ligament of the spine and the fifth cervical vertebra, and at exactly corresponding points on the anterior and posterior walls of the pharynx.

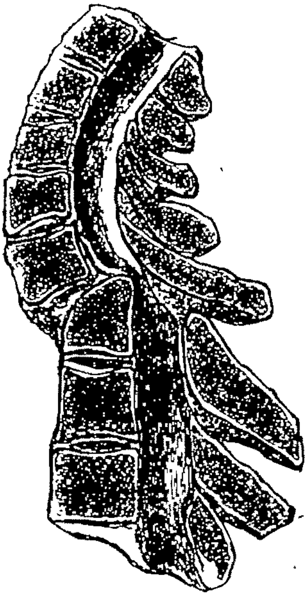

FIG. 4.—Sketch of section of spine in case of J. S., showing fracture-dislocation of the seventh cervical vertebra. From a drawing made by Mr. Moss.

This interesting case is in itself almost a demonstration of the greater part of our table. The injury was quite at the lower part of the dorsal region, and must have at first involved the first dorsal nerves and probably to some extent the eighth cervical. At first we had paralysis only of the intrinsic muscles of the hand and the interossei, but thereafter from day to day we were able to watch the extension upwards of myelitis, picking out muscle by muscle in the following order:—

1. The flexors of the wrist.
2. The extensors of the wrist.
3. The triceps and pectoralis major.
4. The latissimus dorsi.
5. The teres major and subscapularis.
6. The deltoids, flexors of the elbow, and external rotators of the humerus (supra- and infra-spinati) remained until the last.

The dates at which the pronators failed could not be obtained.

We have thus described the seven cases of fracture-dislocation, and shown that in the main they strongly support the above arrangement. It is true that there are some discrepancies, and some gaps still remain to be filled up, notably with respect to the exact position of the nuclei for the pronator muscles; but considering the nature of the investigation, I think I may fairly claim to have established the original proposition, and may hope with some further observations to verify and complete the results. At least I hope that I have demonstrated the interest and importance of a more exact study of the distribution of paralysis in these cases than is usually attempted—an importance which may be especially great in medico-legal investigations.

In order to illustrate the way in which such observations may assist us to arrive at an exact diagnosis, I now bring forward three more complicated cases of spinal injury, and shall endeavour in each to exactly localise the lesion. A similar accurate study would probably clear up and establish a definite diagnosis in many cases now vaguely described as "concussion of the spine."

CASE 8.—R. M., aged 55, a labourer, was admitted to the Manchester Royal Infirmary on April 27, 1887, under the care of Mr. Whitehead. He informed us that about 10 or 11 P.M. on the previous night he had fallen head first down some seven or eight steps, and being able to move his right arm only he had lain as he fell until 7 A.M. I only heard of his admission about 12.30 P.M., and then went at once to the ward with Mr. Collier, then resident surgical officer, and Mr. Iddon, house surgeon. When I first saw him, he presented the following symptoms:—The lower limbs appeared to be quite paralysed, but were said to have been moved once or twice during the morning: they were very rigid: the knee-jerk was much exaggerated and the cremasteric reflexes absent. On respiration costal movement was slight, but quite distinct.

Anæsthesia extended as high on the trunk as the second rib, but was not quite complete. Urine was retained; the penis was not turgid. The condition of the upper limbs was more interesting. The left was completely paralysed, lying by the side. The right was abducted at the shoulder, with the elbow strongly flexed, so that the hand lay above the shoulder with the palm backwards, *i.e.* in a state of pronation, the fingers and wrist being slightly flexed. He could raise and rotate the shoulders, but had no other power of movement in the limb. There was almost absolute anæsthesia of the left upper limb, but on the right side loss of sensation was limited to the ulnar aspect. Both pupils and palpebral fissures were small. The head and neck were markedly bent to the right, and there was a distinctly felt lateral curvature in the cervical region. We now decided that there was a dislocation, probably unilateral, of the fifth cervical vertebra, and, having in view the otherwise inevitably fatal termination of the case, we resolved to attempt immediate reduction. This we did as follows:—The man lying on his back in bed, I seized him firmly by the shoulders, and Mr. Collier grasping the head drew it upwards and to the right, then bringing it over to the left. A slight snap was felt, and the position of the head was rendered more nearly but not yet quite straight. The same proceeding being repeated, a snap was again felt, and the head came into its normal position. At this moment the patient screamed with pain shooting down the left arm, and situated, he said, on the side further from the thumb. This pain lasted only for a minute or two. Within half a minute he flexed his left elbow, and then again kept the limb quiet with the elbow flexed. On again examining the sensation of the limbs, we found partial sensibility on the ulnar side of the left upper, but the radial side remained as before. At the same time we noticed a diffused swelling in the left posterior triangle of the neck, due doubtless to effused blood, which swelling I may at once say gradually subsided without giving any trouble. Finally we adapted to the patient's head, neck, and shoulders, a felt casing, designed to keep them in position. No deformity of the head or neck remained. At 2 P.M., an hour later, the patient could move the elbow and fingers on the left side, and sensation as before was absent only on the radial side of the limb. There was no marked rise of temperature either now or at any subsequent period in the course of the case.

On the following day he passed a large amount of urine, the catheter was not required, and from this time he had no trouble with retention. The urine had a sp. gr. of 1024, contained some albumin and decolorised Fehling's solution, without giving a precipitate; it also contained a large quantity of water. The penis was slightly turgid. There was a good deal of pain between the shoulders, but no other change.

On April 29, the third day, sensation was better in the lower limbs, especially on the right side and in the anæsthetic portions of the upper limbs. The patellar reflexes were less exaggerated than before, and the limbs less rigid. The right upper limb presented as before the symptoms of complete paralysis of motion and sensation below the fifth cervical root: on the left side he could feebly flex and extend the fingers, wrist, and elbow; could pronate and supinate the wrist, and slightly abduct and adduct the humerus. The urine was now alkaline, very fœtid, and contained pus. The optic discs were normal. A day later he had more power in the left upper limb, and anæsthesia was nowhere complete. On the right side he had gained some power of pronating and of extending the elbow, and the area of sensation was extending. The knee-jerks were about normal; the contraction of the pupils less marked. The above figure, taken from a photograph obtained for me by Mr Sidley at about this period, shows the position of the limbs and indicates the marked abduction of the humerus and flexion of the elbow on the right side. From this point it is unnecessary to give a daily record, but it will suffice to note merely a few of the more important changes. Sensation very gradually returned throughout, being preceded, as I have elsewhere shown to be usual, by a

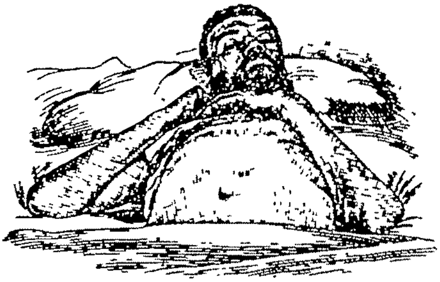

FIG. 5.—From a photograph of J. M., showing the position of the limbs in a case when the fifth cervical nerve-root was injured on the left side only.

sense of tingling: the right lower limb recovered more quickly than the left. Traces of albumin and sugar disappeared from the urine within a week, as did the pus and the alkaline reaction. For a long time he had some difficulty in passing urine, but this gradually passed off. The pupils returned to their normal condition in about a month, the right doing so more slowly, and even as lately as January 1888 being smaller than the left. For some days he had troublesome bronchitis and all movement of the ribs ceased, but it then gradually returned.

The knee-jerks had quite disappeared by May 5, and after being absent for some time gradually returned in the exaggerated form of spastic paralysis which they still retain, and which is more marked on the right side. On May 11 he could move the lower limbs about the bed very fairly. In about six months he could walk. In the upper limbs return of power was equally slow, and there is here a good deal of atrophy: this atrophy and weakness is still more marked on the right than on the left side. In August all the muscles of the left upper limb reacted well to the faradic current, whereas on the right side only the deltoid, biceps, and supinator longus did so; so that on that side the "reaction of degeneration" was present below the fifth root.

After the accident the nails of both hands grew in a markedly clubbed form.

Since he left the Infirmary this man has frequently reported himself to me, and in April last I showed him to the Clinical Society of Manchester. When I last saw him in August 1888, he could walk well, but with the usual spastic gait, and complained only of a feeling of numbness in the lower limbs, abdomen, and hands. His upper limbs are a good deal wasted, but he has very fair power throughout and can do light work, having no difficulty even in such movements as are required for lacing his boots or buttoning his clothes.

In this case I think we may assume that there was a dislocation of the fifth cervical vertebra, and that the traction thereby caused had injured the left fifth cervical nerve-roots at their point of exit from the cord, so as to completely paralyse sensation and motion in the distribution of that nerve, but that the cord itself was not completely crushed, having only sustained hæmorrhages into its substance. As shown in the accompanying diagram, the right fifth cervical root would escape—the cord below this region would be affected by partial destruction (which was worse on the right side)—and the left fifth cervical root would, as already stated, be torn or greatly stretched, so as to suffer temporary total paralysis.

Hence we have on the right side partial paralysis of the upper limb; the deltoid, biceps, and supinator longus escaping, as in the other reported cases, where the fifth root was spared, with retention of sensation on the outer side of the limb supplied by the same nerve. All sensation is lost below this root, and the pupil is smaller in size upon this side of the body than on the other, coinciding with the greater amount of damage done to the cord. This is also evidenced by the fact, that anæsthesia was more marked on the left than on the right side in the lower part of the body, and that the paralysis of those muscles of the upper limb supplied from below the fifth root was more marked on the right side, as was the exaggeration of the tendon reflexes. On the right side also we have the "reaction of degeneration" resulting, whereas on the left it did not do so. On the left side, on the other hand, we find that the movements of reduction, by irritating the overstretched fifth root, caused the muscles supplied by it to contract, but that both motor and sensory paralysis were more marked and enduring in this root than elsewhere. The urinary and other symptoms are compatible with a partial destruction of the cord, and passed off to

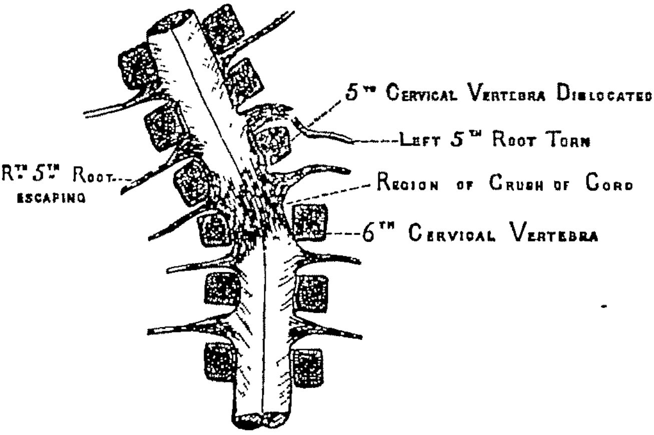

FIG. 6.—Diagram of the supposed nature of the lesion in his case of J. M. The left fifth cervical nerve-root should appear as crushed or stretched rather than completely torn.

a large extent as repair ensued. One point in this case is quite unusual; namely, the early exaggeration of the patellar reflexes, these being as a rule lost, owing to the shock of the injury: whereas here, doubtless from irritation, they were increased;[1] as the irritation subsided, they disappeared, and then at a later stage reappeared in an exaggerated form—the usual result of an old transverse lesion.

CASE 9.—J. A., a bailiff, aged 62, attended as an out-patient under the care of Dr. Ross in December 1886. He told us that nine weeks previously he had fallen about nine or ten feet, and, having held out his arms to save himself, came down upon his hands. For a second or two he felt nothing, but had then great pain shooting down the arms to the hands. His upper limbs then became spasmodically contracted, so that he held them with the elbows to the side and flexed, the hands in front and partially flexed —*i.e.* as he showed us, very much in the position in which a book might be held for reading. This pain and spasm lasted for about a fortnight, since which time they have been gradually abating, and he has now not much pain, and can move the arms in all directions, although they are still weak and he cannot well throw them backwards. The legs have felt cold and weak since the accident.

On examination we found hyperæsthesia, cutaneous and deep, extending across the shoulders from the sixth or seventh cervical spine to the acromia. He had no spinal pain or tenderness. On carrying the arms backwards there was pain which prevented his dressing himself. He had no trace of anæsthesia. Over the hyperæsthetic region was a slight herpetic eruption. All the arm muscles appeared to be fairly strong. The lower limbs felt weak, and the knee-jerk was increased. There were no urinary troubles.

On Jan. 18, 1887, I again examined him more carefully. No local signs of spinal injury could be detected. The hyperæsthetic band was limited above at the level of the sixth cervical vertebra, and below by a line some two inches lower; it extended outwards on either side for about three inches—*i.e.* as far as the distribution of the descending branches of the cervical plexus. Its boundaries were well marked, especially above. The herpetic eruption had almost disappeared, and seemed on inquiry to have been due to previous friction with acetic acid.

On examining the upper limbs, I found fair power in the deltoid, flexors, and extensor of the fore-arm and extensors of the wrist, but marked weakness of the flexors of the wrist and fingers. In the pectorals I found nothing unusual. The hands and flexors of the wrists were wasted to some extent. All the muscles of the upper limbs reacted to moderately powerful faradic currents. No sensory change could be made out in the upper limbs, and there was no jerk on striking the triceps or wrist tendons.

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[1] Since writing the above, I have seen this result in another case.

Examined in a dim light, the pupils were distinctly smaller than normal, the change being more marked on the right side, and they did not dilate on pinching the neck. The legs were well developed and presented no paralysis, anæsthesia, or trophic lesions; but the patient stated that they did not feel so strong as formerly, and were always either cold or burning hot. The knee-jerks were markedly exaggerated; the superficial reflexes absent. The bladder and rectum presented no abnormality.

I saw this patient again on April 28, 1888, when he told me that he was much better than he had been at first; but that he still felt some feebleness of the arms and legs, and that occasionally the legs felt hot or cold. He presented no very marked signs of injury, being quite able to go about and perform all the usual movements with his arms and legs. The knee-jerks were slightly, but not much exaggerated. He had no pain, anæsthesia, hyperæsthesia, or cutaneous eruption. His arms were, however, not very strong, and there was obvious wasting of both triceps muscles. On making an electrical examination, I found that the flexors of the fore-arm required a more powerful faradic current than the biceps, the extensors a still more powerful one, and the triceps could only be got to contract with very strong faradic currents. With the galvanic current cathodal closure in every muscle caused contractions more readily than anodal closure, so that there was no reaction of degeneration. Hence the muscles of the upper limb were partially atrophied and insensitive to the faradic current in varying degrees, the most healthy being the biceps, then the flexors of the wrist, then the extensors, and the weakest being the triceps.

This case would appear to be one of hæmorrhage into the central grey matter of the spinal cord, most severe in the neighbourhood of the triceps nuclei, but extending into those immediately above and below. The flexors of the elbow and the shoulder muscles escaped entirely the destroying lesion, but irritation caused the spasmodic position of the limbs which he at first experienced; similarly there was spasm below the region of the hæmorrhage causing flexion of the fingers and wrist. Hence we appear to have had spasm in two groups of muscles—those above and those below the lesion: viz. above, the biceps, subscapularis, teres major, latissimus dorsi, and pectoralis major; and below, the flexors of the hand and fingers. At the stage when we saw the man this spasm had passed away: the paralytic symptoms were most prominent, affecting the central group of muscles—the triceps, and to a rather less extent the extensors of the wrist and fingers. The region of hyperæsthesia corresponded to the posterior divisions of the central nerves of the plexus. It is notable that we found no sensory affection in the distribution of the anterior divisions, *i.e.* in the central part of the arms, but from various observations I am inclined to believe that the central nerves of the brachial plexus have a very slight distribution in the upper limbs, and doubtless the anterior and posterior bands which they do supply would shortly be vicariously supplied by the upper and lower branches on either side of them. The diagnosis of a central hæmorrhage rather than a meningeal lesion must rest chiefly on the rapid onset of the symptoms. Meningitis could not cause symptoms within a few minutes, and a meningeal hæmorrhage would be less distinctly localised.

Case 10.—R. P., aged 25, a porter, was admitted to the Manchester Royal Infirmary under Mr. Hardie's care on December 19, 1887. He had on the same day been walking along the streets, which were slippery, when he fell at full length on his back. He was seized with a sudden pain at the lower part of the neck, shooting down the arms, and at once sustained complete loss of power in his legs and partial loss in his arms. He was never unconscious.

When examined, he presented severe pain in the lower cervical region, shooting thence down the arms. No deformity of the spine could be felt, but pressure caused great pain between the fourth cervical and first dorsal vertebræ, and pain was caused by pressing the head downwards or moving it from side to side. He lay in bed with the fore-arms folded across the chest, the elbows being a little abducted, the hands prone; abduction and adduction of the humerus were however fairly well performed, as were pronation and supination. Flexion of the fore-arm was not much impeded, but the power of extension was lost. The grasp of his hand was exceedingly feeble, and extension at the wrist resulted from the effort. The flexors and extensors of the wrist were weakened, apparently about equally so. The intrinsic muscles of the hands were very feeble. These symptoms were similar on both sides, except that on the right adduction of the humerus was less powerful than on the left, whereas the grasp of the hand was stronger on the right side, and unaccompanied by extension at the wrist. On the left side sensation was thought to be somewhat deficient in the fingers, but there was no other affection; on the right side it was normal.

The chest movements were feeble, and the legs showed great loss of power, but sensation was unaffected. The knee-jerk and plantar reflexes were exaggerated on both sides. He had no bladder or rectal troubles; the urine was of sp. gr. 1019, acid, containing a trace of albumin, but no sugar. Temperature, 100° 6; pulse 80. Ext. ergot. liq. was administered several times.

Two days later the arms were slightly stronger, the legs decidedly so; the temperature had come down and shortly reached the normal.

On December 27 extension of the fore-arms was still weak, but the other movements much better. He had now ceased to retain the fixed position described above, except at times, and had hardly any pain. Improvement was rapid, and on January 16, 1888, he was discharged cured, except for some slight feebleness of the grasp.

The above account is taken from notes by Mr. Bannister, the house surgeon, as I only saw the case a few days before his discharge.

This is another instance of a small hæmorrhage, mainly in the region of the triceps nucleus—a hæmorrhage confined to the anterior cornua, and affecting only the motor apparatus. It is needless to repeat the evidence, which shows that it was most severe in the region of the triceps nucleus, but extended thence to those of the extensors and flexors of the fingers, and that it was at a rather higher level on the right than on the left side, so that on the former the abductors of the humerus were also weakened.

The distribution of the *anæsthesia* in the above cases is of interest, equally as great as that of the paralysis. In his most interesting and important paper in the January number of 'BRAIN,' Dr. Ross has fully demonstrated the nature of the distribution of the sensory nerves of the upper limb. Regarding the limb in its embryological position, we find that it is projected as a bud from the trunk, the hand being supine and the radius upwards, so that the palmar surface is anterior. In this position the bud carries out with it branches of the anterior primary divisions of the spinal segmental nerves from the fifth cervical to the first dorsal inclusive; and as those nerves maintain in the adult their embryological relations, we have the several roots supplying the limb in numerical order from the radial to the ulnar side. Hence, then, the higher the paralysis extends in the cord through the brachial region, the further will the anæsthesia extend from the ulnar towards the radial side. This arrangement is fully demonstrated by our cases. It is unnecessary to refer again to the distribution which will be obvious to any one who takes the trouble to read the reports, and of which I have already spoken in my previous paper. It will be found that the fifth root supplies the region overlying the deltoid muscle, and the outer aspect of the arm and fore-arm as far as the styloid process of the radius or base of the thumb, and that the eighth cervical and first dorsal supply the little finger and inner side of the hand, fore-arm and arm, the remaining roots providing for the central parts of the limb on both anterior and posterior aspects. It will, however, be found that these central roots supply relatively a much less extensive area than those above and below them. Thus in Case 7 the extension of the anæsthesia was much slower than that of the paralysis, and in Case 9, where the lesion affected the centre of the brachial region, this sensory disturbance was at the end of nine weeks appreciable only in the posterior primary divisions, there being none in the limbs. In Case 10 there was no definite anæsthesia, owing probably to the hæmorrhage being localised to the anterior cornua; the region in which, as I have before shown, it is always most marked.

In referring to certain other points in these injuries, I shall consider the whole of my nineteen cases together.

As regards the *nature* and *seat* of the lesion we have thirteen cases of fracture or dislocation and six of hæmato-myelia, or possibly five of hæmato-myelia and one of meningeal hæmorrhage. The dislocations were thus distributed:—

Between the 4th and 5th cervical vert.	3 cases.
„ „ 5th „ 6th „	„ 5 „
„ „ 6th „ 7th „	„ 1 case.
„ „ 7th „ 1st dorsal	2 cases.

In Cases 1 and 2 of the present paper there were no *post-mortem* examinations, but probably the injury was a fracture dislocation of the fifth vertebra. All the cases conform to the usual type, the upper vertebra being dislocated forwards. In every case which I have examined there was some fracture accompanying the dislocation; the usual condition being either a distinct fracture, or a mere "grinding off" of the anterior superior edge of the lower vertebra. Further injury to the bones may or may not coexist. In two cases which I did not see there was said to be no fracture, but the coincidence is so remarkable that I am inclined to suspect that the above described "grinding off" was unnoted. This fracture is often so slight that, unless a vertical section be made through the affected bones, it can hardly be recognised. The condition is represented in Fig. IV. where such a section was made. In Case 4 only the dislocation was lateral, but as the patient is living the exact condition cannot be ascertained. In the three cases of hæmato-myelia previously reported, the hæmorrhage was situated about the level of the fourth or fifth root, the region which, as I then explained, is most liable to this injury. Of the two cases here given, both were about the sixth root.

It is not always easy to ascertain the exact action of the force which *caused* the injury, but of the nineteen cases probably nine or ten were due to over-extension or over-flexion of the neck (Nos. I., II., VII., 1, 2, 3, 4, 5, 6, and 8).[1] In five cases (III., IV., VI., VII., and 10) a blow or crush of the shoulders was the source of the injury. One (IX.) was a gunshot wound. One (9) was due to a fall upon the outstretched hands, and may therefore probably be regarded as due to jerking of the head forwards. In two the nature of the injury was very obscure—they were both falls, and there was nothing to show how the patient struck himself.

In only five cases was there noted any *local deformity*, and in many of the others its absence was remarked upon: in this connection it must however be remembered that I only saw these cases after they had been laid flat in bed—a procedure which frequently reduces the deformity. In dislocations *local pain* is invariable. As it is frequently suggested in courts of justice that a fracture or dislocation of the spine is always accompanied by pain on pressing downwards upon the head, I have recently paid particular attention to this point; and in five cases of dislocation and two of hæmato-myelia where I sought for it at an early period, I found the symptom once only, and that in a case of hæmato-myelia (10).

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[1] I refer to cases in the earlier paper by Roman numerals, to those in the present one by Arabic numerals.

Hyperæsthesia is a symptom much less commonly found than the text-books would lead us to suppose. Among the fracture dislocations it was only once (Case 5) distinct, and twice (Cases 4 and 6) was slightly marked; in all the others it was absent. In Case IX., a gunshot wound, with probably meningeal hæmorrhage, it was well marked; and in Case 9, one of hæmato-myelia, it was also very distinct. Hence I can see no value in this presence or absence of hyperæsthesia, which is commonly stated to be a valuable diagnostic sign between dislocations and fractures respectively.

The *tendon reactions* were noted at an early period in eight cases of fracture-dislocation, in seven of which they were lost immediately after the accident. In one case (8) they were markedly exaggerated. The loss of these reactions is attributable to the shock of the accident, and in the exceptional cases we must assume that the lesion caused great irritation of those portions of the cord situated below it. In this instance they shortly disappeared and were lost on the eighth day. I had wished to ascertain the period at which the knee-jerks return, but as most of the cases die too early this is not easy to do. In Case 4 return was between the fourteenth and nineteenth days. In Case 8 they were still absent at the end of the seventh week, but I have no further note until the fifteenth week, when they were again very much exaggerated, my note at the time being "the most marked I have ever seen." In Case I. there was no return at the twenty-first day—the date of death—and all the other dislocations died before return was possible. Of the cases of hæmato-myelia we find in VII. exaggeration on the eleventh day, and in all it was found sooner or later; in Case 10 it was noticed immediately. Hence then we can lay down no definite rule as to the period at which spastic symptoms ensue, having every variation from an immediate onset to one delayed for over seven weeks.[1]

The *superficial reflexes* are generally lost below the seat of the lesion. Their condition was noted in eleven cases. In ten they were lost immediately or very soon after the accident. In Case 5 (a woman) the plantar reflex was exaggerated and the abdominal present, but as the patient only survived for forty hours, we cannot tell how long they would have remained so: it is notable that this is the only case with marked hyperæsthesia.

Contraction of the *pupils* and *palpebral* fissures has been already sufficiently dwelt upon: all of these cases illustrate the condition. We have, however, to refer to one point not hitherto noted, namely, the fact that the pupil does not dilate on irritating the skin; the physiological explanation is obvious. I have been anxious to ascertain the condition of the optic discs, and have examined the majority of the cases, although I only find notes in four. In those which were examined, but where there is no note, the condition was certainly normal. In Cases 6 and 8 no change could be detected—during periods sufficiently remote from the accident. In none of the cases which recovered was there any complaint of eye symptoms, so that even without an ophthalmoscopic examination we may assume that there was no optic neuritis. In two cases (4 and 7), however, we find very distinct changes. In Case 4 the discs were on the fifth day ill defined and showed venous congestion, but no further changes occurred during life (twenty-eight days). In Case 6 there was similar haziness and congestion on the third day, and distinct neuritis on the ninth; as death occurred on the tenth, the results could not be followed. I am aware that on such a point the ophthalmoscopic observations of anyone but a specialist are open to doubt, but in the present instances I had the advantage not only of seeing the change occur from the healthy to the diseased condition, but also of having my conclusions confirmed by Dr. Little, who kindly examined the cases for me and whose name leaves no room for doubt.

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[1] I have since seen two cases: one a fracture-dislocation of the cervical region, when exaggerated knee-jerks appeared at once and lasted until death; another of the mid-dorsal region, where there is still no knee-jerk at an interval of six months after the injury.

Some interesting *vaso-motor* effects were observed. In twelve cases of fracture occurring in males priapism was present from the first in eleven and persisted until death. In the twelfth case (8) it only appeared on the second day, and gradually passed off as recovery ensued. The thirteenth case of fracture (5) was a woman, and here I was told by the nurse that there was a profuse vaginal discharge: unfortunately this information only reached me after death, and I was unable to examine the nature of the discharge, but it is at least probable that it was a secretion due to changes analogous to those causing priapism in the male—a condition I do not remember to have hitherto seen noted in any spinal affection. On the other hand, of five cases of hæmato-myelia occurring in males, only one presented any turgidity of the penis, and this one only for about twenty-four hours (Case VI.). This I believe to be due to the vaso-motor fibres being situated near the periphery of the cord, and thus escaping the effects of a central hæmorrhage.

The *skin* was generally dry and warm, even when the temperature was low, but there are several exceptions to this condition. In Case 6, where the right cervical sympathetic was more affected than the left (as indicated by the smaller size of the pupil), the temperature was one degree higher in the right than in the left auditory meatus.

High temperatures were only found in four cases (III., IV., V., and 2), all of which were fracture dislocations high up in the brachial region, and all of which died within forty-eight hours. A remarkably low temperature was observed in Case 8, where the lesion was between the last cervical and first dorsal vertebræ.

The *pulse* was usually slow and almost always full and soft, and in Case 3 presented a very remarkable respiratory rhythm; there being an exaggeration of the normally larger amount of blood expelled by the heart during inspiration.

The *urine* was retained in all the cases of fracture dislocation. In all cases of hæmato-myelia it was either not retained at all (VIII., 9 and 10) or only for a short period (in

VI. for one day; in VII. for four days); whereas in IX., a female with meningeal hæmorrhage, there was prolonged incontinence. This I believe to be due to the same reason as the turgidity of the penis, viz. to the fibres for the bladder being peripherally situated. (See above, and also p. 540 of 'BRAIN' for January 1887.)

Transient *glycosuria* was noticed in two cases (5 and 8), and transient *albuminuria* in two cases (8 and 10); increased density of the urine with excess of urea in Case 7; increased secretion of uric acid in Case 8; and of phosphates in several cases. These results, which are by no means constant and which only persist during a few days, would appear to point to important vascular changes in the kidneys.

In Case 6 there was on the second day excessive hæmaturia, with fœtor of the urine—an instance, apparently, of acute trophic lesion of the kidneys. And it is perhaps worth noting that in this case the bladder had not been relieved for two days, and that the hæmaturia came on shortly after the first use of the catheter.

THE PATHOLOGICAL ANATOMY OF A CASE OF LOCOMOTOR ATAXY, WITH SPECIAL REFERENCE TO ASCENDING DEGENERATIONS IN THE SPINAL CORD AND MEDULLA OBLONGATA.

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AND

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THE patient was a male, aged 35, a bricklayer, who was admitted into St. Thomas's Hospital on May 20th, 1886. He was under the care of Dr. Ord, who has kindly allowed us to use the case.

There are many points in the clinical history of much interest, but for our present purpose a brief account of the condition during life is all that is necessary.

The patient stated that his symptoms began two years previously with weakness of the lower limbs and shooting-pains, and that a little later he found difficulty in walking in the dark. About a year after the first onset of the symptoms the patient began to lose control over his bladder, and five months subsequently, after a severe headache, he had double vision and squint. There was no history of syphilis.

On admission, he could not stand or walk without assistance. There was great inco-ordination of the legs, and some loss of power, chiefly affecting the quadriceps femoris.

There was no muscular wasting. Sensation was much impaired in both feet and legs, but elsewhere on the body it was good. The knee-jerks and the tendon reactions of the arms were absent, as were also the plantar and cremasteric reflexes. The abdominal and interscapular reflexes were said to be present, though the latter was slight. There was no loss of power in the arms and no obvious inco-ordination.

The pupils were small, but the left was larger than the right. On admission, it was noted that they acted to light and during accommodation, but after he had been in the hospital six months the pupils were said to exhibit the typical Argyll-Robertson condition.

Three months later, a week before death, it was stated that there was no reaction either to light or during efforts at accommodation. Occasionally he had diplopia on looking to the extreme right, and it was thought that there was slight weakness of the right external rectus. There was no colour-blindness, and the optic discs were normal.

The condition of the patient until death may be summed up in a few words. He had frequent attacks of pain above the symphysis pubis, sometimes accompanied by rigors and elevation of temperature, and usually by an alkaline state of the urine. Several times it was noted that the urine contained phosphates and pus.

Within a few weeks of his death he had on two occasions a series of epileptiform attacks, probably of uræmic origin. During the last week he had hallucinations and was very noisy. He died on February 23rd, 1887.

Post-mortem Examination was made twenty-two hours after death.—Body fairly nourished. There was no urate of soda in the metatarso-phalangeal joints of great toes; but there was some erosion of the cartilages, especially near the margins. No bony outgrowths. Skull-cap and dura mater natural; venous sinuses contained soft clot only.

Brain.—At the vertex the arachnoid was thick and opaque, and the Pacchionian bodies unusually large. There was general excess of sub-arachnoid fluid over the convex surface; the vessels were moderately distended. The pia mater was readily detached. The optic nerves, chiasma and tracts, the third, fourth, sixth and other nerves at the base seemed healthy.

The left vertebral artery was of normal size, the right about one-third the size of its fellow. There was a small localised atheromatous patch on the basilar artery, but with this exception the vessels at the base were healthy.

The motor convolutions were normal. In the cortex of the left gyrus fornicatus, just in front of the rostrum of the corpus callosum, there was an ill-defined, slightly grey patch, which was possibly an islet of early sclerosis. With this exception there was no sign of degeneration. The central ganglia were healthy. The cerebellum appeared normal.

The bodies of the vertebræ and the dura mater were natural.

Spinal Cord.—Nothing abnormal was seen on the anterior aspect of the cord. On the posterior surface, from the upper dorsal region downwards, the pia-arachnoid was dull-white and thickened, and the sub-arachnoid fluid was in excess. A few small calcareous plates were seen attached to the surface of the arachnoid posteriorly. There were no adhesions between the dura mater and the pia-arachnoid. The posterior roots and ganglia, especially in the dorsal and lumbar regions, were much atrophied, grey and translucent. The anterior roots were natural. The cord was somewhat smaller than normal, but not markedly flattened in front or behind.

Only a few sections of the cord were made in the fresh state. In the lumbar and lower dorsal regions the entire posterior column had a grey, translucent appearance, the posterior horns being also involved. In the cervical region the columns of Goll (postero-median) only seemed affected, but the posterior cornua were very slender.

In the medulla oblongata the fasciculus gracilis and apparently the fasciculus cuneatus on both sides were sclerosed.

The vagi, the right anterior crural nerve, and the right quadriceps muscle appeared healthy.

Larynx, trachea and bronchi contained some blood-stained mucus.

The following were healthy: the lungs, heart, liver, spleen, adrenals, œsophagus, stomach and intestines.

The first part of the aorta was much thickened generally, and exhibited many large patches of atheroma, but no calcareous plates. In the rest of the aorta there were scattered, raised patches of atheroma.

The urethra was healthy. The bladder was much dilated and hypertrophied. Both lateral lobes of the prostate were softened, of a dirty-slate colour, and infiltrated with pus. The ureters, which were dilated to two or three times the normal size, contained some turbid urine. The pelves and calyces were much dilated, a little injected, and contained a few small concretions of uric acid, but no pus. The capsules of the kidneys were much thickened and adherent to the surrounding tissues. The capsules were readily detached; but the surface of the kidneys was irregular and lobulated, and showed some small, round points which were probably minute abscesses. On section, the cortex was irregular and very obscure, but exhibited no suppurative points.

The spinal cord, medulla oblongata and pons were hardened in Müller's fluid and subsequently placed in spirit. In the spinal cord sections were made at the level of each nerve-root from the apex of the conus medullaris upwards. The staining was by the Weigert hæmatoxylin process.

Microscopical Examination. Spinal Cord: at the level of the coccygeal nerve.—Even here, the lowest part of the cord, the posterior columns are much sclerosed. Some of the nerve-fibres on each side of the postero-median fissure still remain. The area of fine nerve-fibres lying in front of the entrance of the posterior nerve-roots (Lissauer's tract) is but little degenerated. The network of fine medullated fibres has disappeared from the posterior horns, and no nerve-fibres can be seen passing from the posterior root-zones to the cornua, as occurs in the normal state. There is an evident excess of fibrous tissue in the lateral columns, especially just in front of the posterior cornua.

Sacral region.—The posterior sclerosis is well marked; but on passing from the lower to the upper part of the sacral region a gradual increase in the number of healthy nerve-fibres is seen in that part of the posterior columns which abuts on the posterior grey commissure. This tract of normal fibres extends backwards as a wing-like expansion, following the contour of the cervix cornu posterioris on both sides. The horizontal fibres which normally pass from the postero-external columns to the posterior cornua are almost absent. In the caput cornu posterioris the substantia spongiosa, especially in its posterior part, contains far fewer medullated fibres than it does normally. On the other hand, the reticulate region of the grey matter immediately in front of the posterior cornu, is quite healthy.

At the level of the third sacral nerve, Lissauer's tract is profoundly degenerated.[1]

(Figs. 1 and 2 represent the gross characters of the lesion in the lower and upper sacral regions.)

Lumbar region.—The degeneration of the posterior columns is greater here than in any part of the sacral region. In the postero-external columns, behind the cervix cornu posterioris, there are two symmetrical areas of fairly healthy nerve-fibres. Throughout the lumbar region, Lissauer's tract is completely sclerosed, and the horizontal radiations from the posterior root-zones into the cornua are absent. In the lateral columns there is diffuse sclerosis, the degeneration being much less advanced than in the posterior columns. The lateral sclerosis does not merely include the pyramidal-tract area, but extends far beyond it, especially anteriorly and towards the middle line.

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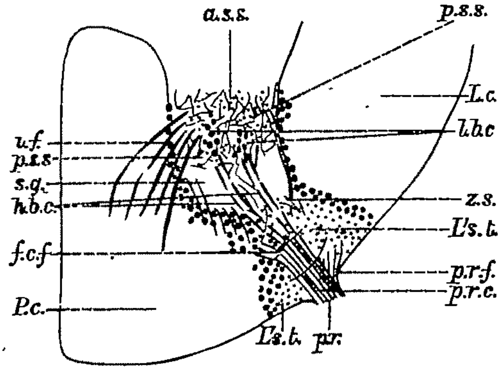
[1] We append a Schema of the constitution of the posterior cornu in the lumbar region according to Lissauer (slightly modified from the original in the 'Archiv f. Psychiatrie,' vol. xvii. p. 377).



Schema of the Constitution of the Posterior Cornu, according to Lissauer.

L.c., lateral column.
P.c., posterior column.
p.r., posterior nerve-root.
p.r.c., coarse fibres in *p.r.*
p.r.f., fine fibres in *p.r.*
L's.t., tract of Lissauer.
a.s.s., anterior region of substantia spongiosa.
p.s.s., posterior region of substantia spongiosa.
s.g., substantia gelatinosa.
z.s., zona spongiosa.
l.b.c., longitudinal bundles of large fibres.
h.b.c., horizontal bundles of large fibres.
f.c.f., fine commissural fibres.
i.f., irradiating fibres from posterior column.

Dorsal region.—Between the ninth and tenth dorsal nerves the sclerosis in the posterior columns reaches its greatest intensity. The few fibres that remain occupy an area on each side behind the cervix cornu posterioris. There is great diminution in the number of the medullated fibres in the posterior cornua, and also in the posterior vesicular columns, except just in the front of the cell-groups. The fibres which are mainly affected in Clarke's columns are those situated internal to the cells and which pass to the posterior white commissure. There is no obvious alteration in the appearance of the cells in the posterior vesicular columns; but after a careful comparison with healthy sections the large nerve-cells seem fewer than normal, the small cells presenting no diminution in number.

In the substantia spongiosa of the posterior cornua the horizontal fibres are quite absent, whereas there are a considerable number of fibres running vertically. The medullated fibres of the posterior commissure have suffered severely. The horizontal radiations from the posterior root-zones and the horizontal fibres in the substantia spongiosa of the posterior horns are absent.

The formatio reticularis is quite healthy. The lateral columns, however, are much sclerosed, and the change, as in the lumbar region, spreads far beyond the pyramidal-tract area and reaches up to the periphery of the cord. About the level of the seventh dorsal pair the change in the lateral column extends forwards, and gradually becomes larger and more defined on passing upwards. At the level of the eighth dorsal nerve-root the posterior vesicular columns and the cells contained in them become more obvious, and the fine vertical nerve-fibres normally surrounding the columns can be distinguished. On the other hand, there is still a marked paucity of horizontal fibres both in Clarke's columns and in the substantia spongiosa of the posterior cornua, and the horizontal radiations of the posterior root-zones seem to be destroyed.

In the lower dorsal region Lissauer's tract is completely degenerated, but in the upper dorsal region it is less affected; and the anterior part seems normal.

In the posterior columns more nerve-fibres are now visible, and at the level of the sixth dorsal nerve-root they are collected into groups symmetrically placed in the postero-external columns (see Fig. 3). On passing upwards, these symmetrical areas gradually increase in size, and the nerve-fibres contained in them are arranged more compactly. The situation and dimensions of these islets at the level of the third dorsal nerve are represented in Fig. 4, and Fig. 5 exhibits them at the level of the eighth cervical nerve-root.

Cervical region.—Below the level of the eighth cervical nerve the posterior pair of these symmetrical areas is made up of nerve-fibres of large size, but now a small number of fine fibres can be distinguished.

In the lower half of the cervical enlargement the groups rapidly increase in size, and at the level of the sixth cervical nerve the anterior and posterior groups on each side become continuous. This condition of things is well seen at the level of the fifth cervical nerve (Fig. 6). The comparatively healthy area in each posterior column, formed by the fusion of the anterior and posterior groups, is roughly wedge-shaped, and its base, which corresponds with the free surface of the posterior column, passes some distance into the postero-median column. The grey matter above the level of the seventh cervical nerve-root is healthy, and it must be specially noticed that the fine medullated fibres in the posterior cornua show no signs of degeneration. About the middle of the cervical enlargement Lissauer's tract seems normal throughout.

The change in the lateral columns is seen at the periphery of the cord as a well-defined area, which gradually increases in size on passing from the lower to the upper cervical region (compare Figs. 5, 6 and 7).

Medulla Oblongata.—The change in the lateral columns can be followed from the highest cervical region into the medulla, where it occupies the position indicated in Figs. 8 and 9. It is wedge-shaped, with its base directed outwards and extending to the periphery, the apex lying between the antero-lateral nucleus in front and the substantia gelatinosa behind. The deep arciform fibres are not degenerated, and they can be seen coursing circumferentially through the sclerosed area in Fig. 9.

On passing upwards in the medulla the tract becomes less distinct and lies more posterior, until finally it passes into the restiform body. The difficulty in tracing the tract is due partly to the fact that it is divided obliquely by the ordinary transverse sections, and partly to the fact that it is no longer a compact area, but becomes broken up and scattered among other fibres.

The fasciculi graciles, like the postero-median columns throughout the cord, are densely sclerosed, the degeneration being traceable as high as their nuclei. The fasciculi cuneati are much less completely sclerosed, and here again the change can be followed up to the level of the nuclei.

REMARKS.—There are some points of pathological interest in this case to which we wish to direct special attention. The lateral columns exhibit evident degeneration from the coccygeal region upwards, the change in the lumbar and lower dorsal regions being diffuse and extending far beyond the pyramidal-tract area. In the upper dorsal and cervical region the degenerated area becomes more defined, reaches up to the periphery of the cord, occupying a position midway between the anterior and posterior nerve-roots. This sclerosed area, when traced upwards, is found to be directly continuous with the wedge-shaped patch of degeneration in the medulla oblongata, lying in front of the substantia gelatinosa (Fig. 8). Now this is the site of the direct cerebellar tract, and the position usually assigned to this tract in the spinal cord is not degenerated in our case.[1] This being so, it is reasonable to infer that the so-called "antero-lateral tract" in the spinal cord is simply the more ventral or anterior portion of a tract proceeding to the cerebellum, in other words, of the direct cerebellar tract.

We may remark, in passing, that in normal sections this sclerosed area in the medulla oblongata is occupied almost exclusively by nerve-fibres of large diameter, similar to those composing the direct cerebellar tract and the so-called antero-lateral tract; and that, moreover, these large nerve-fibres in the medulla become oblique on passing upwards, enter the restiform body and so reach the cerebellum.

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[1] On referring to Fig. 6, it will be seen that there is slight degenerative change behind the antero-lateral tract on one side, limited to the periphery. This may be secondary to the thickening of the pia mater. In no other part of the cord was there the slightest suspicion of change in the direct cerebellar tract.

With respect to the change in the lateral columns in this case, it is interesting to note that the lesion appeared to bear a relation of an inverse nature to that in the postero-external columns. As the latter became more and more occupied by healthy nerve-fibres, the degenerated areas in the lateral columns become denser and larger. It seems to us very probable that there is a supplementary relation between these two regions of the cord. The anatomical characters in our case suggest that certain fibres of the posterior nerve-roots, after an upward course of longer or shorter extent, pass forward into the lateral column of one or both sides. If this be so, it would appear that some of the fibres of the posterior nerve-roots ascend directly to the cerebellum.

It is difficult to pronounce positively that the change in the lateral columns is of a systemic nature, but the probability is that such is the case. This view is borne out by the condition of things seen in the medulla, where the deep arciform fibres pass quite unaffected through the sclerosed area (see Fig. 9).

It is held by some observers that the antero-lateral tract finally enters the antero-lateral nucleus in the medulla oblongata. This view has been adopted by Dr. Tooth in a recent communication,[1] and in support of this he quotes an observation by Westphal.

Dr. Tooth observes that "the cerebellar element is seen in the restiform body, the antero-lateral remaining where it was: higher still the same tract is very small, evidently close on disappearing. One thing seems certain, viz. that these fibres do not pass to the cerebellum *viâ* the restiform body." Our observation, however, is not in harmony with this view. It is interesting to note, in relation to this point, that in our case the fibres of the degenerated area in the medulla entered the restiform body at a higher level than is usually the case with the direct cerebellar tract.

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[1] "Note on the Ascending Antero-lateral Tract."—'St. Bartholomew's Hospital Reports,' vol. xxiii. See also vol. xxi., "A Contribution to the Topographical Anatomy of the Spinal Cord." Dr. Tooth here admits the possibility, in the case narrated, that the antero-lateral tract passed with the direct cerebellar tract into the cerebellum.

Dr. Gaskell has shown that there is a sharp distinction, both anatomical and physiological, between the small and large nerve-fibres in the anterior roots. It would be of the greatest interest to obtain more information respecting the small fibres in the posterior nerve-roots. In our case it will be noted that these fibres, as well as those of the larger kind, are degenerated in the greater part of the cord, and that Lissauer's tract, which is the continuation of these small fibres, is also profoundly affected. From the facts at our command, we are not in a position to afford any information concerning the function of these fine nerve-fibres of the posterior roots.

Our conclusions may be summarised as follows:—(1) The antero-lateral tract passes to the cerebellum through the restiform body, occupying in the medulla the position of the direct cerebellar tract. (2) The antero-lateral tract is the more ventral or anterior part of the direct cerebellar tract. (3) The fibres which compose the antero-lateral tract, at any rate in part, proceed from the posterior spinal nerve-roots, and these latter are thereby brought into direct relation with the cerebellum.

ADDENDUM.

We had originally intended to deal with the subject of ascending degenerations more fully, and with this view Fig. 10 was added to the plate. The case was interesting, both clinically and pathologically; but a brief account is all that is necessary at the present time. In 1876 the patient was in St. Thomas's Hospital, suffering from angular curvature and spasmodic paraplegia. He regained almost complete power, and when he was admitted again in 1883, it was noted that he was "able to walk perfectly." After his death, which occurred a little later, the cord was found profoundly degenerated. Below the point of compression the lateral sclerosis was very marked. Just above, as seen in Fig. 10, there was also change in the lateral columns, but to a less intense degree. The degeneration partially involved the pyramidal-tract area. It may be noted here that the pyramidal-tract area normally contains fibres of different

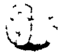

Fig 1

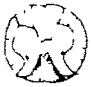

Fig 2

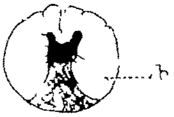

Fig 3

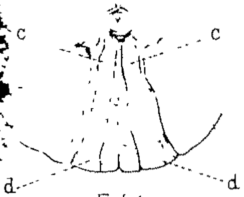

Fig 4

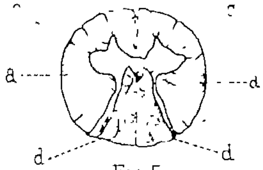

Fig 5

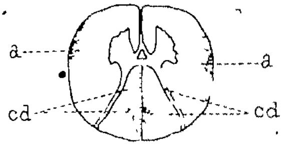

Fig 6

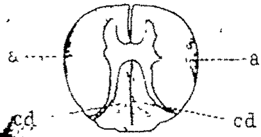

Fig 7

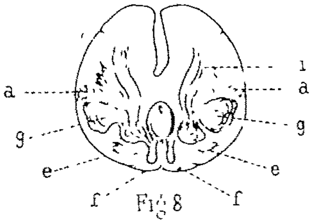

Fig 8

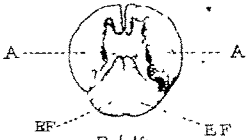

Fig 10

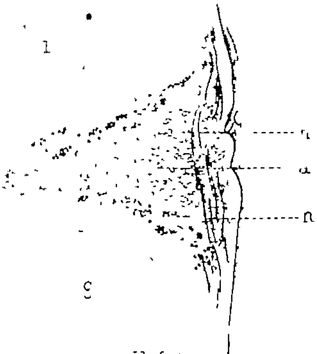

Fig 9

size and probably of different function, and there is little doubt that certain of these fibres, as seen in the fig., degenerate in an upward direction.

EXPLANATION OF PLATE.

Fig. 1. Transverse Section of Cord in Lower Sacral Region.
Fig. 2. „ „ „ Upper „ „
Fig. 3. „ „ „ at 6th Dorsal nerve-root.
Fig. 4. „ „ „ 3rd „ „
Fig. 5. „ „ „ 8th Cervical „
Fig. 6. „ „ „ 5th „ „
Fig. 7. „ „ „ 2nd „ „
Fig. 8. „ „ Medulla Oblongata opposite the upper pyramidal decussation.
Fig. 9. The degenerated area in Fig. 8 enlarged.

All the figures, except 4 and 9, are twice the size of the mounted specimen, from which they were outlined with the camera lucida. Fig. 4 is magnified ten times, and Fig. 9 thirty times.

a = sclerosis in the lateral column, concentrated and well-defined.
b = diffuse sclerosis in the lateral column.
c and *d* = the anterior and posterior islets of nerve-fibres in the posterior columns.
e = degeneration in fasciculus cuneatus.
f = degeneration of fasciculus gracilis.
g = corpus Rolandi.
h = fibræ arcuatæ profundæ.
i = nucleus antero-lateralis.

Fig. 10 (see Addendum). Dorsal region of the cord above the point of compression in a case of angular curvature (magnified two diameters). The stippled parts indicate the healthy tissue, which in the mounted specimen was coloured dark by the Weigert process.

E F = degenerated posterior column. A = degeneration in lateral column.

ON THE EVOLUTION OF THE CENTRAL NERVOUS SYSTEM OF VERTEBRATA.

BY J. BLAND SUTTON, F.R.C.S.

Hunterian Professor; Assistant-Surgeon, Middlesex Hospital.

IN this Journal (January 1888) I attempted to show, from pathological and embryological data, that the intimate relations which exist between the spinal cord and alimentary canal, may be explained on the assumption, that the central canal of the nervous system may be regarded as a modified portion of bowel, and the principal facts adduced in support of that opinion were as follows:—The continuity of the gut and central canal of the cord in the embryo; their similarity in mode of development and correspondence in point of time; the relation of the sympathetic ganglia to the gut and cord; the association of certain malformations of the central canal of the cord with malformations of the alimentary canal; and the relatively large size of the cord in lower vertebrata.

Nearly three years ago I ventured to state, "notwithstanding that we were absolutely in the dark, concerning the significance of the central canal of the cord, it was justifiable to regard it as an obsolete canal, basing this opinion on the ground that it behaved itself like functionless ducts and passages in general, and at times formed cysts."[1]

It gave me considerable satisfaction to hear Dr. Gaskell express, before the Anatomical Society of Great Britain (assembled at Cambridge in June 1888), the opinion, that the tube which primarily represents the central nervous system in the vertebrate embryo, must be regarded as a disused segment of the primitive alimentary canal. Dr. Gaskell had arrived at this conclusion from quite a different mode of

[1] "On the Conario-hypophysial Tract," &c.

working to that adopted by me: it is of interest and importance that two men working independently and in different directions should arrive at fundamentally the same conclusion, although, as may be anticipated, there is some difference in minor details.

As far as I am aware, nothing in any way approaching this somewhat startling conclusion has ever been suggested; exception, perhaps, should be made to the unsatisfactory attempt of Owen,[1] who supposed that the gullet of ancestral vertebrates passed through the third ventricle, and that the space from the pineal to the pituitary bodies, by way of the infundibulum, represented this primitive or invertebrate (cephalopodic) gullet.

Invertebrates are distinguished embryologically from the majority of vertebrates by the fact that, in the early stage of development, they undergo a process termed invagination, whereby a portion of the outer wall of cells is depressed in such a way as to give rise to a double-walled cup (Fig. 1). This is the gastrula stage, and represents temporarily the double-layered condition permanent in many invertebrata. The hollow, G, in the gastrula is the archenteron, the primitive digestive cavity; the cells lining it constitute the endoderm

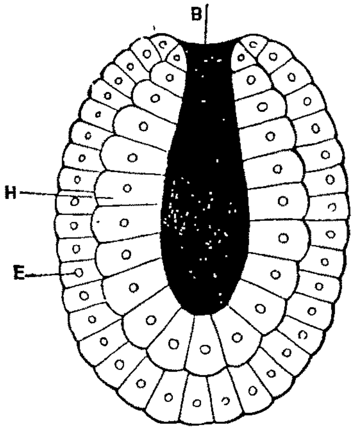

FIG. 1.—A diagram representing the gastrula. E. Ectoderm (epiblast); H. Endoderm (hypoblast); G. Archenteron; B. Blastopore.

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[1] 'Journal of the Linnean Society,' 1883, p. 131.

(hypoblast); the outer wall is the ectoderm (epiblast); the two layers become continuous at the edges of the blastopore. It should be mentioned that the gastrula stage occurs in amphioxus as in invertebrata, and has been detected in the ova of some fish and amphibia.

Ingenious attempts have been made to identify the occurrence of something, which in the higher vertebrates corresponds to the gastrula stage of invertebrates, for the intimate connection of the embryological processes throughout the animal kingdom strongly suggests the occurrence of the gastrula in a modified form, even in the highest mammals. Up to the present time nothing satisfactory has been advanced in this direction. The views which I shall now endeavour to express were forced upon me whilst investigating the mode of origin of those singular ovarian tumours known as dermoids.

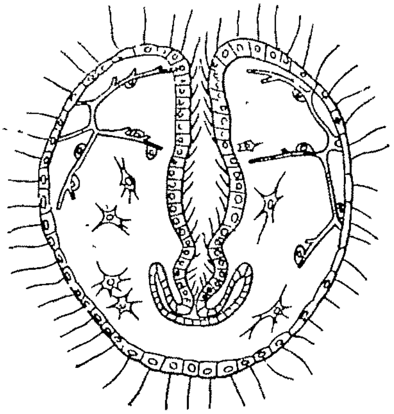

FIG. 2.—Transverse section of the embryo of *Echinus miliaris* to show the origin of the pleuro-peritoneal cavity (cœlom) from the primitive digestive cavity. (From Hertwig, after Selenka.)

It has long been customary to regard the cœlom (pleuro-peritoneal cavity) as a dependency of the vascular or lymph system, but the investigations of the Hertwigs[1] show this view of the matter to be untenable; it is on the contrary a derivative of the archenteron. Each pleuro-peritoneal cavity, for they are at first quite separate, arises as a diverticulum from the central cavity of the archenteron as represented in Fig. 2.

[1] '*Die Cœlomtheorie.*' Jena, 1881.

The central portion persists as the permanent alimentary canal, whilst the lateral diverticula converge ventrally and coalesce to form a common chamber. A transverse section of the embryo of amphioxus is represented in Fig. 3, in which those portions of the primitive digestive cavity which become abstricted to form the body-cavity, are clearly indicated, whilst Fig. 4, representing a Polygordius larva in transverse section, shows the relation of the diverticula to each other when the abstriction is complete. It may perhaps be more conclusive to indicate the relation of these parts to each other in a vertebrate. In Fig. 5, a transverse section through the head of an embryo dog-fish is represented, and the relation of the medullary folds to the gut and cœlom is clearly shown. This view of the nature of the cœlom is of great importance from a pathological stand-point, because it clearly indicates that the lining of the peritoneal cavity is a true

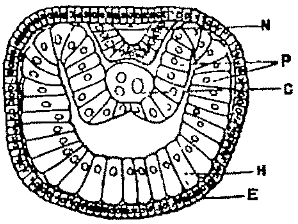

FIG. 3.—Transverse section of the embryo of Amphioxus, showing the abstrictions of the primary alimentary canal which form the cœlom. (After Kowalevsky.) N. Medullary folds; C. Notochord; H. Hypoblast; E. Epiblast; P. Cœlomic epithelium.

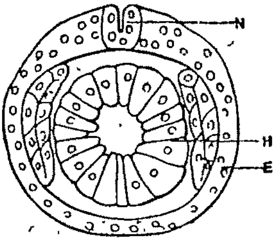

FIG. 4.—Transverse section of a *Polygordius* larva, to show the relation of the alimentary canal and the rudiments of the cœlom. (After Hatschek.) N. Medullary folds; H. Hypoblast; E. Epiblast.

epithelium of hypoblastic origin. Embryologically it is equally important, as it tends to show that the peculiar manner in which the cœlom arises in most mammals, viz. as a schizocœle, must be regarded as a secondary or modified form of invagination, and represents only a part of the gastrula stage of invertebrata.

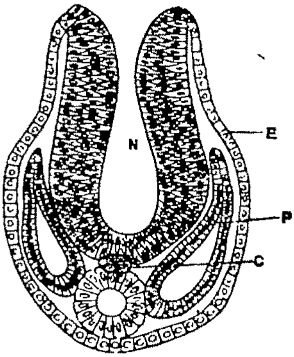

FIG. 5.—Transverse section through the head of an embryo dog-fish, showing the parts representing the gastrula cavity. (After Balfour.) N. Medullary folds; E. Epiblast; P. Cœlom, or pleuro-peritoneal cavity; C. Notochord.

We may now turn our attention to the space in the gastrula which lie between the blastopore and the wall of the tube we have just been considering. In vertebrata, one of the most conspicuous structures in the early embryo is the notochord, concerning which a great deal of discussion has been carried on at various times as to which of the germinal layers it is derived from. Morphologically, its most conspicuous features are its close association with the alimentary canal, and relation

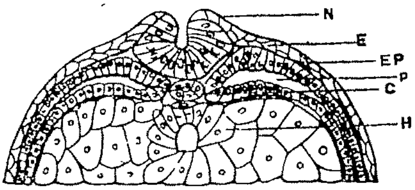

FIG. 6.—Transverse section of a three days' embryo of *Triton tæniatus*, showing the cœlom arising as a schizocele. (After Hertwig.) N. Medullary folds; E. Epiblast; H. Hypoblast; C. Notochord; P. Cœlom.

to the spinal cord.[1] If we glance at the gastrula of *Echinus miliaris* we find that by approximating the edges of the archenteron at the point N, we should produce a thickening, and divide the cavity into a dorsal and ventral section, the part below corresponding to the bowel and cœlom, and the parts on the dorsal aspect would represent the medullary folds of the vertebrate; now occlude the blastopore, and we at once give rise to an arrangement of parts corresponding in transverse section to the disposition of parts seen in the early vertebrate embryo, and in longitudinal section would furnish us with the fundamental U-shaped tube from which I started in the previous communication. Such a relation of parts is shown in the longitudinal section of the embryo of the amphibian *Bombinator igneus* (Fig. 7).

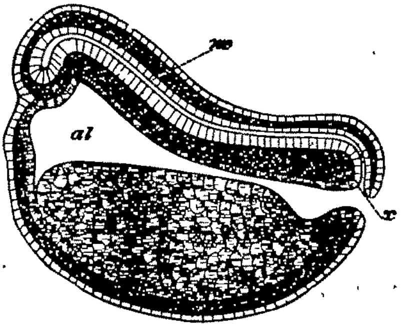

FIG. 7.—Longitudinal section of an embryo of *Bombinator igneus*, to show the relation of the neural and alimentary canal. (Modified from Gœtte.) *n c.* Neural canal; *a l.* Alimentary canal.

This view tends to show that the up-growths known as the medullary laminæ, and the down-growths forming somatopleure and splanchnopleure represent a modification or an abridgment of the invagination process so universal among invertebrata. Among the advantages which follow upon this view of the origin of the central canal of the cord and brain is this one—it absolutely removes an objection which a few are disposed to urge against the intestinal origin of the canal in question, viz. that its epithelium is epiblastic, whereas that lining the gut is hypoblastic. In its simplest form, the hypoblast is that portion of the epiblast which after invagination lines the archenteron; following up this view of the matter, all the parts arising as modifications of the archenteron in higher and more complex animals, must naturally be regarded as hypoblastic. Thus, according to this view, the epithelium of the central canal of the nervous system from the infundibulum of the third ventricle to the extremity of the cord, that lining the neurenteric passage, the alimentary canal as far upwards as the junction of œsophagus with the pharynx, the epithelium of the trachea, bronchi, &c., the epithelium of the pleuroperitoneal cavities, and that lining the organs derived therefrom, must be regarded as of hypoblastic origin.

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[1] I omit for the present any reference to the sub-notochordal rod, which is more intimately connected with the gut than the notochord.

His' discovery that the cells which make up the medullary folds in the embryo are not, as is usually taught, metamorphosed into nerve-cells, but form the sustentaculum—the myelo-spongium—of the nervous axis is an important fact in support of the intestinal origin of the spinal cord, and has been used with considerable skill by Gaskell in this direction.

Of course it is as yet impossible to discuss this theory in connection with the many side issues it opens up, but this is certain, it will throw considerable light upon many dark problems of pathology, and especially one which has been regarded "as linked with some of the profoundest mysteries of organic life," viz. ovarian cystomata.

THE EPILEPTIC PAROXYSM.

BY JAMES OLIVER, M.D., F.R.S. (EDIN.).

THE whole molecular world, organic as well as inorganic, exists in a perpetual state of trepidation. In the former, life is the manifestation of an inherent power possessed by its molecules of adaptation to surrounding circumstances, and it is the complete failure of this capability which renders the animate at once an inanimate object. As we ascend the scale of animal life, we witness the gradual evolution of a heterogeneous and highly instable representative structure, the nervous system, a medium of communication not only between every molecule of the body, but between every ultimate tissue of the organism and its environments, and through which, by a process of action and reaction, equilibration is maintained. In the body itself and its surroundings, there are many factors for ever at work, which tend to avert molecular stability, influenced as this is by those changes affecting the elasticity or density of the structure. If the elasticity of any body be materially augmented, we shall forthwith detect a corresponding increased proneness to be influenced by and radiate molecular disturbances; whilst any increase, on the other hand, in its density must necessarily be accompanied by a diminution and limitation of this proclivity, bearing, as these two qualities—elasticity and density—do, a direct antithesis to each other. Although the causation of epilepsy, exciting as well as predisposing, is still a question wrapt in obscurity, there can exist no doubt in the scientific mind that the manifestation of the disorder is the result of some occult change which has arisen in the whole or some part of the cerebro-spinal system. In man, the structural arrangements of the nervous system are so complex, and the functions so entangled, that a satisfactory and withal comprehensive description of the epileptic paroxysm is rendered necessarily difficult. The most typical attacks, however, are those in which the discharge beginning in the highest nerve centres descends in a more or less markedly progressive and rapid manner, and invades the whole representative structure. The mode of onset may and frequently does vary, not only in different individuals, but in the same individual at different times; we may therefore encounter every possible variation according to the part of the nerve structure primarily disturbed, the completeness or incompleteness of its molecular instability and proneness to radiation. When a fuse is applied to any part of a gunpowder tract of like elasticity and density throughout, the molecular disturbance may travel in any or every direction. It is possible, however, to so alter in parts the density of this highly instable compound as to intercept effectually the molecular wave in its passage, and thus limit its power of propagation. This simile, to my mind, reveals well the true character of a nerve discharge, and the manner in which it may spread in those with an epileptic predisposition.

Attendant Sensorial and Intellectual Disorders.—Deriving, as we do, all knowledge of our own existence, as well as that of an outside world, through the medium of our special senses, including the tactile, of which all the others are highly organised modifications, I propose to include in a common description the disturbances manifested by, and attributable to, changes arising either in the centres receiving or in those translating impressions. Here, as elsewhere, the discharge may be more or less confined; the causal instability being limited, the surrounding structure remaining unaffected by the apparently spontaneous molecular trepidation, further radiation is prevented. The whole question of sensorial and intellectual epileptic disturbance is one wrapt in obscurity, fraught with complexity, and surrounded with difficulty, yet teeming with interest, and from a medico-legal point of view worthy of our most serious consideration.

Hitherto loss of consciousness was and still is by some considered an essential sign of epilepsy; it is, however, a variable accompaniment of the manifestations of this disorder. In many confirmed cases I have witnessed exactly the same epileptic phenomena in the same patient, at different times associated with complete or incomplete loss of consciousness, or wholly unattended by any sensorial or intellectual disturbance whatever.

When the mental change is evinced, it is not necessarily an early symptom. Many patients retain for a more or less indefinite period of time, after the initial disturbance, a full knowledge of their own bodily state and everything that happens around them. The epileptic discharge, it is true, begins usually in the highest part of the cerebro-spinal system, and descends; it may, however, originate primarily in any part of the nervous tract, and radiate more or less irregularly. In the most marked and severe epileptic attacks, loss of consciousness is invariably complete, and is ever, I believe, in such the earliest manifestation of the paroxysm. The discharge under such circumstances is as a rule so rapidly transmitted, that the abolition of consciousness and the evolution of the motor and other allied disturbances are apparently simultaneous. The period during which there is this total interference with the reception and translation of impressions appears to, and is spoken of by, the patient as "a blank." The individual in a state of epileptic unconsciousness may or may not retain his equilibrium; he may, without any warning whatever, fall suddenly on the ground in a helpless and passive state; or he may maintain his posture, and evince no further outward manifestation other than a somewhat strange and vacant, yet withal characteristic stare and fixity of the eyes. In yet another set of cases we witness the evolution of thought, more or less coherent and intelligible, and the performances of actions more or less complete and difficult of attainment, with a total suppression of consciousness. With the initiation of unconsciousness, the individual frequently falls, sustaining at times severe bodily injuries, even fractures and dislocations. Here, doubtless, the onset is so sudden and shock-like in character, that there results a complete inhibition of the motor centres and consequent cessation of the ever-existing tone with total muscular relaxation and loss of equilibration. In the individual who retains his position, the discharge may travel and affect the whole representative structure of the special senses, or be confined to one or more of these special sense-centres. As a rule, the disturbance in such occurs in the centre appreciating and translating impressions, and not in that receiving them. It may, however, occur in one or both, and we may encounter every possible variety. There may be complete and very transient loss of eyesight, with or without accompanying auditory change—the patient retaining the power of hearing, but being at the same time wholly incapable of comprehending and defining the impressions conveyed to the auditory centre. The auditory tract may remain undisturbed, the individual comprehending and retaining what may be said, and yet there co-exist with complete cessation of the functional activity of the visual centre a total inability to express in spoken words thoughts resulting from these auditory impressions. Mental phenomena may, however, exist and be revealed, whilst consciousness itself is in abeyance, the individual under such circumstances being a wholly irresponsible agent. Many of the actions performed during this state appear more or less automatic in character; often they are most complex, and may be exhibited apart from any evident or necessary motive.

During a state of epileptic unconsciousness I have frequently witnessed the patient run forward with much impetus, as though impelled by some irresistible power. In one case, the individual coming in contact with a bed which happened to impede the course, was thrown violently on the floor, performing a somersault, and landing on the other side of the bed. This patient sustained a severe bruise of the left hip by the fall, and continuing unconscious for several seconds afterwards, would have succeeded in recovering the erect position and rushing on again, had I not prevented her by physical force. These movements may by some be considered as post-epileptic or even hysteroid in character; that, however, they were an accompaniment and not a sequence of the epileptic state, I have no misgivings. Many patients display an awkward propensity for undressing, and it is often difficult to prevent them from throwing off every vestige of dress on the body. This peculiarity may be a concomitant of the minor attack with mere loss of consciousness, or a truly post-epileptic phenomenon exhibited after a severe paroxysm. In one case which came under my notice—that of a female—the patient always undressed, and sometimes even got into bed when such was possible, and this whether in her own house or that of any friend or stranger. The importance of a knowledge of such facts from a medico-legal point of view cannot be overrated. The act may result from a feeling of impending illness (prior to the attack), for in the majority of our automatic movements the cell and fibre arrangement is so perfect, that the least stimulus may serve to call into action, and evoke unconsciously, with machine-like regularity, the associated train of events. The act of undressing has doubtless, through habituation, become more or less automatic. In some cases, at least, it appears to be totally unconnected with any feeling of indisposition, for one lad I interrogated whilst so undressing as to why he did so, replied, "I am going to work;" and this patient, having already thrown off his coat and waistcoat, had unlaced and taken off his boots, and was about to take off his trousers, when he recovered consciousness and re-dressed, retaining no knowledge whatever of what had transpired. Another patient, the subject of severe as well as minor epileptic attacks, had during the occurrence of a minor attack started to prepare tho table for tea; he arranged the dishes with wonted regularity—a duty assigned to him some weeks previously—and was astonished, on regaining consciousness, when it was told him what he had done. Some years ago I remember a female patient telling me that, in her slight fits, she habitually—irrespective of the time of day—placed some tea in the tea-pot with hot water, and placed it by the fire, but never did she at any time remember anything of the act, nor any reason for so doing.

Our feelings, thoughts, and actions, are usually conscious; that, however, they are not invariably so, appears more than problematical. To say the individual feels without knowing he feels, is somewhat paradoxical; it cannot, however, be more so than to say the individual thinks and acts without the knowledge of thought or action, tenets which we must admit.

Although the whole structural arrangement necessary for the reception and conduction of impressions from the periphery to the higher centre is intact and functionally active, yet because the impressions so received fail to be appreciated by the organism, we say the feeling does not exist. It is not the feeling which does not exist, but the consciousness of the existence of the feeling which is non-existent.

The Cry.—The epileptic cry is, comparatively speaking, an infrequent accompaniment, but an early manifestation of the paroxysm. It is uttered in the aggregate—so far as I have been able to judge—only in about 5 per cent. of the fits. In some cases it is never noted, whilst in others it is an occasional but never a constant association. It appears to be a mere casual motor coincidence, dependent upon a state of more or less complete inflation of the lungs at the time of the initial nerve disturbance. The cry, when emitted, is very characteristic but difficult of imitation, and is a wholly unconscious phenomenon, as the patient never at any time retains any knowledge of its utterance. Sounds more or less articulate in character, and associated with the consciousness of a developing aura and consequent fit, are commonly uttered, and are most probably the result of fear or, in some few cases, apparently of pain.

The epileptic cry is an invariable manifestation of a general seizure, sudden and severe, the molecular change affecting the whole representative nerve-structure in an incalculable period of time. The muscles of the chest and vocal apparatus act in concert through habituation, the resulting automatic centre being located in the highest part of the motor nerve tract, and represented on the surface of one or other hemisphere, according to individual peculiarity in the neighbourhood of the centre for speech.

The epileptic cry, as far as my observation goes leads me to believe, is especially noted in right-handed individuals, when the nerve discharge, judging from external manifestations, originates in the left hemisphere rather than when, in the same individual, the molecular disturbance primarily affects the right cerebral lobe. In left-handed patients, again, I have invariably noted its association with an initial molecular discharge in the right hemisphere. The cry is produced by an associated action of the muscles of the chest and vocal apparatus, whereby the air expelled from the lungs is forced through the narrow glottis. It is a true part of the fit, and may be evinced by patients equally, whether asleep or awake, at the time of onset of the attack.

Motor Disturbances in the Limbs.—In the most characteristic epileptic attacks the motor spasm, so far as the limbs are concerned, appears at first in one or other upper extremity, as the case may be, the initial manifestation being always in that toward which the head is primarily deviated. It then spreads in a somewhat regular manner to the lower extremity of the same side, to the lower extremity of the opposite side, and finally attacks the remaining upper limb. The march of retrogression is, as a rule, in the inverse order of progression, the spasm disappearing first in the group of muscles last affected, and continuing longest in those primarily disturbed. It is but seldom, however, that we witness this uniformity in progression and retrogression, the nerve structure being so complex and so irregularly liable to change, as to allow of almost every conceivable variation.

The convulsion itself is usually divided into two stages, the tonic or initial, and the clonic or sequential. Here again we rarely find the regular association, and may often detect the simultaneous existence of these two muscle states and every possible intervening gradation in the different limbs.

Experimentally, if a stimulus of regular and never-varying quantity traverse the nerve tract of a muscle, no change in its structural state, as evidenced by contraction, will result. In order that a muscle may be thrown into contraction, the force or quantity of the stimulus must vary, and according to the rapidity with which the waves of molecular motion follow each other, so do we develop a tonic or clonic spasm more or less distinct. The tonic state results if the waves of excitation are uniform, and follow each other with such rapidity that ere the influence of each preceding impulse has finally disappeared, a new wave of motion has already been transmitted. Clonic convulsion, on the other hand, is the outward manifestation of impulses, or groups of impulses, transmitted to the muscle, but separated by more or less distinct intervals, which allow of the muscle returning after each excitation to a state of comparative inactivity.

Initial tonic convulsion, eventually replaced by clonic, is commonly observed in pure functional or so-called idiopathic epilepsy; whilst initial clonic spasm, on the other hand, is a common feature of epileptiform disorders dependent, as a rule, on some coarse cortical lesion of the cerebrum. Clonic convulsion, when a sequel of tonic spasm, apparently depends for its development upon a somewhat exhausted state of the still highly instable nerve-cells. Its frequent, though not invariable co-existence, however, with the venous turgescence resulting from interference with the respiration, renders it highly probable that the impregnation of the blood with carbonic acid may aid in determining clonic spasm through increasing the density of the disordered nerve-tissue. Tremor, more or less limited in extent, is frequently noted as a motor manifestation of the epileptic paroxysm. During the fit and under certain circumstances I have been able to develop this trepidation, especially in the lower limbs, by merely straightening the leg already slightly bent by a feeble yet maintained tonic contraction. I have also witnessed the spontaneous development of this variety of spasm or clonus, when the leg slightly bent at the onset of the paroxysm has become straightened and somewhat stiff during the fit itself. Whilst the character of the convulsion may vary, so may the extent and manner in which the muscles are involved; and this in the majority of cases without any very apparent cause. In a few cases with distinct cerebral or spinal lesion I have noted a definite alteration in the character and evolution of the motor phenomena in idiopathic epilepsy. Some years ago I saw a girl, epileptic from childhood, in whom the convulsive seizure had always been general, affecting all four limbs. At the age of ten this patient during a fit suddenly developed a complete hemiplegia (left), the result apparently of a hæmorrhage into the right cerebral lobe. In all attacks hereafter the convulsive phenomena were confined to the right half of the body. The girl is now fourteen years of age, and although she has acquired a fair amount of volitional power over the left arm and leg, the convulsion never spreads to this side.

Although prior to the occurrence of the hæmorrhage into the right hemisphere, the left arm and leg had always evinced motor disturbance, they now remain perfectly quiescent throughout the fit. Here the cerebral lesion is the undoubted cause of alteration in the paroxysm; its mode of operation, however, is at present difficult to determine. In another case I witnessed the rare association of epilepsy with paraplegia, the result of pressure from Potts' disease in the lower dorsal region of the cord. It is more than likely that in this case the epilepsy was truly idiopathic and cerebral, and wholly independent of any cord lesion. The curvature had existed from childhood, and gave rise to no material inconvenience until the age of forty-five, when complete paraplegia resulted. He regained the wonted power in the legs in the course of twelve months, and it was not until one year later that he was the subject of his first epileptic fit. The attacks thereafter recurred more or less regularly every three months, always in the night and during sleep. At the age of fifty-two, seven years after the first attack of paraplegia, he came under my notice, having again lost completely the power in both lower limbs. Gradually he began to amend, and on the day previous to an epileptic seizure I had noted that he was able to move feebly and very imperfectly the left leg, foot and toes; on the right he had no power whatever. During the fit, which was typically epileptic, the head was drawn from side to side alternately; both upper limbs were at first the seat of tonic and afterwards of clonic spasm. The left leg, at first straight and somewhat rigid, manifested during the existence of the clonic spasm in the upper extremities a general tremor, whilst the right leg throughout evinced no change. Later on, having recovered almost completely power in both legs, all four limbs alike were the seat of convulsive disturbances during the epileptic fit. It is more than probable that, in this case, the pressure on the lower dorsal part of the cord determined the freedom from motor disturbance of the right leg, and the trepidation instead of clonic spasm of the left.

Epilepsy, purely idiopathic in character, is often found co-existent with infantile or adult hemiplegia. The association may be a mere casual coincidence. In many such cases the motor manifestation is confined to the hemiplegic side, and the fact that there is frequently no intellectual derangement whatever is indicative of an epileptiform rather than an epileptic disorder, and points to some coarse cortical lesion as the cause of the chronic motor spasm. The length of time which may elapse between the occurrence of the hemiplegia and the epileptic or epileptiform manifestations may vary. In one case which came under my notice twelve years had intervened, and here the convulsions were invariably general, and not limited or confined to the hemiplegic side. The whole question is one wrapt in obscurity, for it is quite possible that, apart from the hemiplegia, this lad might have been the subject of idiopathic epilepsy. The position of the limbs during the paroxysm may vary in the same as well as in different attacks. One or more of the extremities may be unaffected, and occasionally, although rarely, the motor evolution may be crossed, the spasm being located in the upper extremity on one side and in the lower on the other. When three limbs are invaded, it is more commonly noted in one upper and the two lower than in the two upper and one lower. Sometimes one or more limbs may be flexed and one or more may be straight. In one case the upper limbs were invariably raised above the head during the attack, whilst in another they performed a to-and-fro wing-like motion. If the patient during an approaching paroxysm hold anything in the hand, the object is tightly grasped whilst the tonic state exists, but is invariably dropped with the onset of the clonic convulsion.

Flexion is a common posture assumed, not only by the upper but also by the lower limbs. In the upper, while flexion predominates the arm is usually adducted and drawn across the front of the body, the elbow and wrist are more or less markedly flexed, whilst the thumb is bent in on the palm of the hand and the other fingers, too. When the lower limb is similarly involved, the thigh is bent on the abdomen and the leg on the thigh, whilst there is an associated extreme dorsal flexion of the foot. With strong flexion in the limbs the head is often bent forward on the chest, and so powerful and sudden is this initial force, that I have seen a patient thrown out of bed with violence on the floor. In some rare instances I have witnessed a dominating extension of the lower limb, associated with well-marked plantar flexion of the foot so extreme as to make the foot and leg almost straight. Occasionally under such circumstances there is noted a slight disposition to arching of the back.

Micturition.—During the fit there is frequently involuntary passage of urine. This by some authorities is considered a pathognomonic sign of epilepsy; as, however, I have occasionally witnessed the association of this phenomenon with convulsive seizures, typically hysteroid in character, it cannot be viewed as an infallible symptom. Although urination is often a concomitant of the severe epileptic paroxysm with total loss of consciousness, yet the act is one which may be manifested apart from the combination, either with intellectual perturbation alone or mere motor spasm without loss of senses. In one case which recently came under my care, the patient, occasionally the subject of fully developed attacks of epilepsy, frequently experienced a mere trepidation of the nature of a general fine clonus of the right leg. In such minor attacks, and without any intellectual disturbance whatever, the bladder invariably expelled its contents,—the patient retaining throughout a full knowledge of the act, but being wholly unable to prevent it. I have witnessed the bladder empty itself during simple epileptic unconsciousness unaccompanied by any motor manifestation. In a lad, the subject of minor as well as severe epileptic attacks; the minor, consisting in complete loss of consciousness and slight tremor of both upper extremities, especially the right, was invariably associated with expulsion of the vesical contents. This patient never fell during the minor attacks; but if standing, when so affected, maintained the erect position. In some cases the aura of an approaching paroxysm is the feeling of a desire to urinate, and if the act can be forthwith accomplished, the sensation passes off and no further epileptic manifestation ensues. If from surrounding circumstances the bladder cannot be emptied, the sensation may occasionally pass off, but more commonly determines a fully developed fit, in which the vesical contents are expelled. In some few cases there is apparently a hyper-secretion of urine, the water passed immediately after the attack being paler than usual, and of unusually low specific gravity. This anomaly is probably comparable with the enormous secretion of saliva which is occasionally noted, and to which I shall presently draw attention. On three occasions I have detected a slight trace of albumen in the urine after a paroxysm, but never any trace of sugar.

The emptying of the bladder during a fit apparently depends on the state of distension of this viscus at the time of onset. In some cases it appears to result from an inhibition of the bladder sphincter. As a rule, however, it is effected by a direct contraction of the bladder muscles, aided by the superincumbent pressure exerted by those of the abdominal walls.

Erection of Penis and Emission of Semen.—In severe attacks of epilepsy I have witnessed on several occasions erection of the penis, but never at any time ejaculations of semen, nor have I been able to detect after the fit any evidence of its emission. It is possible, however, that should the circumstances favourable for the act exist, emission may result. Erection appears to result from a general involvement of the body structure, as I have never noted its presence in mild attacks.

Defæcation.—The involuntary passage of fæces is an act of very rare occurrence during the epileptic fit, as I have only once noted it in the many hundreds which I have witnessed. In this case the copious evacuation which resulted leads me to believe that the mere tonic contraction of the body muscles as a whole determined the expulsion of the contents of the rectum, apart from any special contraction of the intestinal tract or inhibition of the sphincter muscle.

Salivation.—A copious flow of saliva is a frequent accompaniment of the epileptic paroxysm, and is most commonly noted in those fits which are preceded by a distinct epigastric aura. As a rule the secretion is thin and watery, characters exhibited by that produced by excitation of the chorda tympani nerve, or associated with a distinct feeling of nausea. In several cases the saliva has literally poured out of the mouth, its downward flow into the stomach being prevented by the existence of œsophageal spasm, to which I shall presently draw attention. If the tongue has been bitten, the saliva may be tinged with blood. In a few rare instances I have witnessed a markedly sanguinolent discoloration of the secretion, and yet have been unable to detect any evidence whatever of laceration of the tongue. Under such circumstances the blood is probably the result of a transudation from, or a direct rupture of the blood-vessels situated somewhere in the pharynx.

The secretion of saliva during the epileptic fit appears to be of a purely nervous character, and wholly independent of any direct alteration in the blood-supply of the glands. Increased blood-supply is ever a necessary concomitant of augmented functional activity, but what I mean to infer is that the alteration in the vascular state of the gland is not the initial cause of the hyper-secretion, but merely an essential association. The excitation is probably purely central or cerebral, and not truly reflex, and is apparently unconnected with motor disturbance even of the jaw. I have remarked its presence during a distinct pallor, as well as marked flushing of the face.

Abortion.—In a few rare instances I have noted perfect immunity from epileptic attacks in females during gestation; as a rule, however, this physiological state interferes in no way with the recurrence nor character of the paroxysms. Women who suffer from epilepsy are invariably most prolific, and it is somewhat astonishing that although the whole body structure appears to be more or less invaded by a severe paroxysm, the uterus, then in a state of gestation, is seldom so involved as to expel its contents. The epileptic fit itself I have never known to be the initial factor in producing abortion. The accident, however, occasionally happens, and is commonly the result of some direct injury sustained during the fall.

Vomiting.—Emesis I have never witnessed during the epileptic seizure, although I have in a few rare cases noted its occurrence before and after the fit. The symptom is ever a most dangerous one, as food is apt to pass into the glottis, and bring about a fatal result. When we remember that salivation is a frequent concomitant of the paroxysm, it is surprising that vomiting never occurs. In order that the contents of the stomach may be expelled, the cardiac orifice must remain patent, and this probably never happens during a fit. Contraction of the gullet and cardiac end of the stomach is apparently produced before the gastric organ itself becomes invaded or affected by the superincumbent pressure of the diaphragm and abdominal walls. In a few rare instances, patients who during a meal have felt the usual epigastric aura common to them before a fit, have informed me that they have then occasionally experienced some difficulty in swallowing, a fact strongly in favour of early spasm of the gullet.

Vascular Changes.—Palpitation is often a precursor of the epileptic seizure (minor as well as severe), and in some cases it even constitutes the distinct aura or warning of an approaching paroxysm. During many fits in the male and female alike I have detected a marked increase in the frequency of the pulse, reaching, though rarely, as high as 120 or 140 per minute.

After the cessation of the attack, it gradually lessens in frequency and returns to its normal rate. Augmentation of the pulse is more commonly observed than diminution; in a few rare instances, however, I have found a pulse averaging as a rule 76 per minute reduced during the actual fit itself to 48. In many cases I have remarked no apparent or rather appreciable change, whilst on one occasion only did I note intermission in a pulse which at other times was perfectly regular.

In some cases there is either a marked pallor or a distinct flushing of the face, indicative respectively of contraction and dilation of the blood-vessels; occasionally, however, during the same attack, I have observed an alternation of these two vascular states. During a severe paroxysm the face, irrespective of its initial colour, is invariably livid, because of the interference from muscle spasm with the pulmonary circulation. The cyanosis gradually lessens, and the face re-assumes its normal colour as the clonic spasm becomes more completely interrupted.

During severe fits, hæmorrhages, more or less extensive, may result. In the skin, these usually partake of the character of petechiæ, and are most commonly detected on the neck, the eyelids, and external ears, especially the lobes. On one occasion I witnessed a distinct and, comparatively speaking, profuse hæmorrhage from both internal ears. Not infrequently I have noted extensive sub-conjunctival hæmorrhages in one or both eyes, which underwent during the process of absorption the usual changes incident to such. Occasionally, as I have already remarked, rupture of blood-vessels occurs in the pharynx or œsophagus.

Respiration.—In minor epileptic attacks, with mere loss of consciousness and slight flushing of the face, I have frequently observed a marked acceleration of the respiration. As a rule, however, in the typical attacks, there is interference with the respiratory movements, and signs more or less distinct of asphyxia result from a fixation of the respiratory muscles and spasm of the glottis. In many cases, the asphyxiated state becomes so marked, that death seems inevitable.

The state of the Reflexes.—During the epileptic fit there is total abolition of all reflexes, the pupil fails to respond in the usual manner when exposed to a strong light; the conjunctiva, when touched, fails to elicit a reflex contraction of the orbicularis; and, when favourably tested, the plantar surface of the foot when tickled, fails to produce its wonted contraction of muscles.

Finger Snapping.—Finger snapping is an odd gesture, yet I have once witnessed its manifestation during the epileptic unconsciousness. It is supposed to be a sign indicative of contempt; whether there had been any such idea in the mind of my patient prior to the exhibition of the above peculiar phenomena I failed to elicit.

The state of the Pupils.—During the actual fit itself the pupils are, as a rule, widely dilated; on two separate occasions only have I witnessed marked contraction, a condition which must be considered as very rare. In a lad aged eight, the subject of minor as well as severe and general epileptic attacks, I had the opportunity of witnessing several paroxysms. During and after the minor fits in this case, it was difficult, nay utterly impossible, to elicit with certainty whether unconsciousness was complete or but partial. I believe there was mere intellectual disturbance, the result of limitation of the epileptic discharge to the left cerebral lobe, deranging the higher centres of this hemisphere alone. In the minor attacks he was totally unable to speak, yet appeared not wholly unconscious, but seemed to comprehend, although somewhat imperfectly, what was said to him. The sole muscle manifestation of the paroxysm was a twitching of the orbicularis of the right eye, associated with a dilation of the pupil of the same side, whilst that of the left eye was unaffected throughout the attack. In this case the dilated pupil was on the side towards which the head would probably have been primarily deviated, and away from that hemisphere, the seat of initial discharge. Associated activity of the two hemispheres of the brain is the rule, yet it is possible, and the above case somewhat favours the acceptance of the theory, that dual and independent action is probable. In this lad, the dilation of the right pupil in the minor attacks may be considered as comparable with the dilation which nominally results when an individual is directed to look into space. The pupillary change in this case suggested to me the possibility of my being able to derive some valuable information from the pupil states—so constantly associated with epilepsy, and to which I shall presently refer—in determining approximately the seat of initial cerebral disturbance.

Dilation of the pupils, as is well known, occurs during violent muscular effort; it is, however, a noteworthy fact, that this state of pupil is a concomitant of the minor attack as well as the severe epileptic paroxysm. Pupillary dilation was at one time considered to be dependent on a contraction of the blood-vessels of the iris. Now were the old theory admissible, that contraction of the blood-vessels of the brain, as manifested by a pallor of the face, is the initial change in the epileptic fit, dilation of the pupils, as a result of contraction of the blood-vessels of the iris, might be feasibly maintained. During ordinary blushing the vessels of the retina and the brain have been seen to dilate; it is more than problematical, therefore, that the vessels of the iris are similarly affected. If, however, the pupillary state were the result of alteration in the calibre of the blood-vessels circulating in the iris, we should expect to find dilation of the pupil associated with epileptic pallor, and diminution in size co-existent with the initial flushing. Although contraction of the pupil has been noted during the fit, the condition is extremely rare, and marked dilation is the rule, whether there be manifested an initial pallor or flushing of the face. The pupil state in epilepsy appears to me to be wholly independent of the sympathetic influence, also totally unconnected with any cord change whatever—and is most probably of a purely cerebral origin.

In many cases of epilepsy I have frequently remarked, during the period of immunity from attacks, the existence of a permanent inequality of the pupils. I have also, it is true, occasionally observed an inequality of the pupils in individuals of apparently good health; but the common association of this phenomenon with epilepsy makes it somewhat problematical that it may be a direct result of the proneness to this chronic functional disorder. In some cases, I have noted that the pupils, apart from any attack, are ever widely dilated, and either do not react at all to light or but feebly, the nature of the contraction at the same time being wavy and uncertain. The pupil under such circumstances displays a marked proneness to dilate again, the contraction produced being most fleeting and transitory, which otherwise in good health would continue so long as the exciting cause lasted. When the pupils are unequal, the least effort at accommodation may determine equality, the inequality only reappearing with the state of comparative rest. In testing the reaction to light, I have frequently remarked that whilst the pupils may in conjunction react well to this excitation, separately the one more dilated will be found to contract with less certainty and less markedly than the smaller, and at the same time manifests a great tendency to return forthwith to its pre-existing state of dilation. The iris itself is a highly organized structure, and as the epileptic may be considered as epileptic even to the finger tips, it is quite possible that this protoplasmic veil in the eye participates in the general structural instability. So far as my own observation goes, a careful scrutiny of those cases with inequality of pupils reveals no constant information

INHIBITION.

BY CHARLES MERCIER, M.B.

OF all the Functions of the Nervous System none is so mysterious in its nature or so obscure in its working as that of inhibition. The difficulty of comprehending the method of its action appears to me to have been greatly enhanced by the circumstance, that the most striking and palpable instance of the process, and the one that first arrested attention, is the case of the inhibition of the action of the heart, which is probably an unusual and exceptional form of inhibition. Thus it has happened that the classical example of the process is one that cannot readily be compared with others, nor can we readily argue from this to other instances, and so we are deprived of a powerful leverage.

In order to gain a clear concept of the true nature of inhibition we must go down to the bed-rock of the fundamental properties and powers of the nervous system, and regard it as an apparatus for the storage and expenditure of force. If we regard each cell as a reservoir in which force is stored, and each fibre as a channel by which force is carried, we start from a position which no one will nowadays impugn, and which affords us a secure foundation on which to build an hypothesis of the nature of inhibition.

The first course to be laid down on this foundation is the fact, also undoubted, that the expenditure, or release, or emission, or escape, of force from the nerve cells, never occurs continuously, but always in intermittent, interrupted outbursts. Whether we regard the process in the aggregate or in detail, this fact is apparent. Every part of the body has its periods of activity and its periods of repose. That is to say, to every part of the body force is distributed by the nervous system, at some times in abundance, and at other times in driblets. Every muscle has its intervals of contraction and its intervals of rest. That is to say, to every muscle force is occasionally distributed in large quantity, and occasionally in small. Furthermore, if we look at the intimate composition of each muscular contraction we find that it is similarly constituted. For each tetanic muscular contraction is made up of a large number of simple contractions following each other very closely; that is to say, the current of force which causes each muscle to contract is not a continuous current, but an interrupted current—a succession of separate shocks. So that we arrive at this conclusion:—that the force which is expended by the nerve cells is released, not continuously, but in an interrupted current—in a succession of waves, or rather, more strictly speaking, of bubbles, for there is every reason to suppose that each delivery is entirely separated from its neighbours.

This interrupted delivery of force from the nerve-cells is very remarkable, very significant; and if I might venture to express the opinion, has hardly yet received sufficient attention. Its great significance consists in this, that while the *delivery* of force is interrupted, the *storage* or accumulation is undoubtedly continuous. For while the delivery of force is due to the fall of atoms into simpler combinations, the storage of force is effected by the rebuilding of atoms into combinations that are more complex and unstable. This building up of complex molecules is an instance, and a part, of the function of nutrition, a function which, while it varies in activity from time to time, never, as far as we know, ceases altogether. Hence it appears that while force is continuously being stored in every cell, yet it is delivered not continuously but only from time to time; and when delivered, the delivery is not a continuous stream, but an interrupted bubbling over. The question arises, How is this interrupted delivery to be accounted for?

If we look round the world of physical phenomena, we shall find that there are several other instances of a continuous accumulation, both of matter and of force, being broken up into an interrupted delivery. A continuous stream of gas poured into a liquid escapes as an interrupted series of separate bubbles. Similarly a continuous stream of water falling through the air is broken into an interrupted series of separate drops. The continuous pressure of a stream against a water plant causes, not a steady bending, but a waving to and fro of the leaves. The continuous pressure of the steam on the lid of a kettle causes the lid to rattle up and down with an interrupted vibratory movement. So, too, the continuous tension of electricity on the prime conductor of an electric machine escapes in an interrupted series of separate sparks.

In all these cases, and in every other case in which a continuous accumulation is broken into an interrupted delivery, we find always the same result produced by the same conditions. And the conditions are the opposition of a continuously accumulating force to a resistance that is constant. The continuous accumulation of the gas at the end of the pipe is opposed to the constant pressure of the water, and we get a series of bubbles. The continuous increase of the velocity of the falling water produces a continuously increasing pressure of the air against it, which is opposed by the constant resistance of the weight of the water. The tension of the leaf stalk, increasing continuously as it is bent, is opposed to the constant resistance of the stream of water, and we get a series of rebounds. The continuous accumulation of the pressure of steam in the kettle is opposed to the constant resistance of the weight of the lid, and we get a series of puffs of steam escaping. The continuous accumulation of electric tension on the prime conductor is opposed to the constant resistance of the air, and we get a series of sparks.

It appears therefore reasonable to advance, as a working hypothesis, that the intermittent delivery of force from the nerve-cells in which the force continuously accumulates, is owing to the opposition of a constant resistance, as in the many other cases in which a similar effect is produced. The state of things supposed by this hypothesis will involve certain other consequences besides the intermittent delivery of force, and if we find that the facts are in accordance with the necessary consequences of such an arrangement, we may proceed with some confidence still further to utilise and apply the hypothesis.

If we take the rattling kettle off the fire and put it on the hob, so that we bring the contents from a boil to a mere simmer, we find that we stop the rattling at once, but that every now and then the lid gives a jump and allows a puff of steam to escape. By taking it off the fire we have caused it to "go off the boil," that is to say, we have diminished the rapidity of the production of steam, we have diminished the rapidity with which the tension accumulates, and, the resistance still remaining constant, we have diminished the rapidity with which the outbursts of steam succeed one another. Similarly, if we turn the disc of the electric machine more slowly, the sparks remain of the same intensity, but they succeed one another at longer intervals. With a slower accumulation of tension, a longer time is consumed in arriving at a sufficient strength to overcome the resistance, and consequently the outbursts are farther between. But since the resistance remains constant, the strength that has to be arrived at to overcome it remains constant, and each outburst is of precisely the same intensity as the others.

Is there anything analogous to this state of things in the operation of the nervous system? Do we ever find that the evidence of the outbursts of force from the nerve cells tends to show that these outbursts, while retaining their usual intensity, occur more slowly? I think such evidence does occur. The effect of each separate outburst of force from the nerve cells is to produce a single simple contraction of the muscle. Normally these simple contractions follow one another so fast that they are all fused together into a continuous tetanic contraction; but if they follow one another more slowly they will not be so completely fused together. The vibration which is so fine and rapid as to be imperceptible, as it gets slower will get coarser, and will become perceptible. So that instead of a continuous muscular action there will be a vibratory or tremulous muscular action. In short, there will be *tremor*. If the hypothesis be true that the nervous discharge occurs by the continuous accumulation of a tension against a constant resistance, there ought therefore to be tremor whenever the rate of accumulation of tension is diminished. And what are the circumstances under which tremor occurs? It occurs in fatigue; that is to say, after long continued discharge has exhausted the nerve cells of their usual surplus of tension, and after great demands have been made not only on the amount of energy stored up in the cells, but on the process of storage, which has had to keep pace with an unusual demand. The process of storage being continuous, while the process of expenditure is intermittent, the former process is of course and of necessity slower than the latter; hence if expenditure continues, it must at length exhaust all the energy actually in store, and further demands will have to be supplied from hand to mouth, as it were, by the simultaneous process of storage, which as we have seen is normally slower than expenditure. So that when nerve cells have been discharging for some time, the rate of accumulation of tension in them must diminish; and it is when they have been discharging for some time that tremor occurs. Tremor also occurs in exhausting illness, when the general process of nutrition all over the body is weakened, and when the whole bodily condition indicates a lack of intensity of the nervous discharge. In paralysis agitans the whole nervous system does its work badly; there is great and general loss of nervous vigour; that is, there is evidence of slackened accumulation of tension in the nerve-cells. And in paralysis agitans there is tremor. In drunkenness there is tremor, and tremor is worst at the time that every symptom points to the lowest ebb of nervous energy—in the early part of the day.

So far, observed facts corroborate the hypothesis, that the nervous discharge occurs on the breaking down of a constant resistance. We have supposed the resistance to remain constant while the rate of accumulation of tension varies. But suppose the resistance varies in magnitude, what will be the result? Returning to our illustration of the electrical machine, we find that when resistance is increased by increasing the distance that the spark has to travel, two results occur. The tension must reach a higher point before it can overcome this increased resistance; and if the handle be turned at the same rate, then in order to reach a higher point, the tension must have a longer time to accumulate. So that one effect of increasing the resistance will be to increase the intervals at which the shocks are given out—to render slower the succession of shocks. Also, since the tension must overcome the resistance before the discharge can take place, and since the resistance is increased, the tension at the moment of discharge must be increased. So that as the resistance is increased, the shocks of discharge become stronger but wider apart. As we increase the resistance of the air by increasing the distance that the spark has to travel, we increase the magnitude of the sparks and the loudness of their report, but we render their succession slower. As we increase the resistance to the escape of steam from the kettle by putting a weight on the lid, we cause the lid to make bigger jumps, but the jumps follow one another more slowly than before.

In the case of the nervous system we cannot alter the resistance at will, and indeed our only means of judging of increased resistance would be by noticing a concomitant variation in the strength and the rapidity of the simple contractions; so that we are in danger of arguing in a circle. But this we can do: we can see if there is any condition in which the magnitude of the simple contractions bears, while varying, a direct proportion to the varying magnitude of the intervals between them; and if such a concomitant variation is found, we may justly argue, first, that it completely harmonises with the hypothesis with which we started, and second that it is difficult to conceive any other disposition of forces that would so completely and satisfactorily and simply account for all the phenomena.

The concomitant variation in the magnitude and rapidity of simple contractions is found in the course of almost every epileptic fit. In an epileptic fit, beginning in the face, for instance, the opening spasm is "tonic" in character; that is to say, the movement of the part affected is even and regular, like an ordinary natural movement. The eyes turn to one side, or the corner of the mouth is raised, with a movement so gentle and natural, that but for what follows, we should not know that it was part of an epileptic fit. The next thing is that the eyelids or cheek, as the case may be, begin to quiver with a fine vibration. The simple contractions are beginning to show separately, and the spasm is manifestly stronger. As the fit progresses, the fine vibration becomes coarser and coarser, and at the same time the spasm becomes stronger and stronger, producing greater and greater contortions. By the time the limbs have become involved, the spasm has become markedly clonic in character, and of great violence. Now, as the fit passes off, the several shocks come more and more slowly, but assume a greater and greater intensity, until at length a condition of complete and universal relaxation and flabbiness separates from one another the shocks, which, coming now at the rate of only one or two in a second, are of tremendous violence, and seem as if they would break the very bones. At last one terrific universal spasm, coming and going with the electric suddenness that characterises a simple contraction, ends the fit.

So far as the concomitant variation in the strength and rapidity of the shocks can support the hypothesis of nervous action here advanced, the phenomena of such a fit as that described, which is a very common one, do corroborate it completely. If explained by the hypothesis, the phenomena suppose, of course, that the resistance to the discharge gradually increases from the beginning to the end of the fit; and without speculating as to how this increase takes place, it appears extremely probable that it actually occurs. And if it do occur, then we draw this important conclusion: that in the balance of tension and resistance which we suppose to exist in the grey matter, not only does the rate of accumulation of tension vary, but the amount of resistance also varies from time to time. That the accumulation of tension varies both in health and in disease has already been demonstrated; and although the only variation of resistance that we have been able to identify has been a morbid variation, yet, on the general principle that morbid variations are but exaggerations of normal variations, there can be no reasonable doubt that, if the hypothesis is true at all, it is true that both the rate of accumulation of tension and the amount of resistance to its discharge are subject to variation within the limits of health.

Here, then, we appear to have got an inkling of the way in which inhibition may possibly be produced. If all nerve-cells are furnished ready to hand with a resistance to their discharge which is variable within the normal, then it is conceivable and possible that inhibition may be due to an increase of this resistance up to and beyond the limit of the tension attainable by the cells. There is just a trifle of evidence in favour of this view. Supposing inhibition to be effected in this manner, and supposing in any given case a great increase of resistance to prevent a given discharge; and supposing further a sudden increase of tension up to and beyond the increased resistance; the result will be an outbreak of great violence—of greater violence than if no inhibition had been exercised—for it will take place in the teeth of a greater resistance. I think that experience, to some slight extent, enforces this view. If we have tried hard to stifle a laugh or a yawn, and it at length breaks out in spite of the inhibition, it is more violent than if we had at first given way to it; and the same is surely true of an outbreak of temper.

Leaving now the purely physical aspect of the subject, let us turn to the physiological side, and see what evidence can be obtained of the way in which inhibition occurs.

Supposing the nerve cells to be grouped into clusters, each of which, with its fibrous prolongations and intervening grey matter, exercises a specific influence over a specific part of the body, and is called a nerve-centre, then the most elementary and obvious fact, and at the same time the most significant fact, in connection with the action of nerve centres is that they are not continuously and equably active, but alternate between activity and rest; and if active, their activity may vary much in volume and intensity. The initiation of activity of a nerve-centre is easily accounted for. Its equilibrium is upset, its discharge is started, its activity set going, by the impact upon its elements of a current of force arriving from without,—arriving either directly from the external world through the organs of sense, or indirectly, through the intermediation of other nerve-centres. But how are we to account for the arrest of the activity? If it is suggested that the centre subsides spontaneously by virtue of its inherent internal organisation to a state of rest whenever the stimulus to activity ceases, the answer is that, while there is no evidence in favour of such a structure, there is this powerful argument against it, that it is opposed to the most fundamental laws of physics. The first law of motion is surely as true of nerve-centres as of planets—that a body at rest will continue at rest, *and if in motion will continue to move, ... unless acted on by some extraneous force.* The natural inherent tendency of a nerve-centre that has begun to discharge is to go on discharging until it is utterly exhausted. There is no mechanism discovered, I will venture to say no conceivable mechanism, internal to the centre itself, which will cause the centre to spontaneously cease discharging, and yet leave it ready to discharge again on slight provocation. Once set going, it must, unless arrested by some external agency, continue to discharge until emptied of its store of force. But this never occurs normally in the healthy organism. Practically we never become so exhausted by performing any given action that it is impossible to perform it once more. A single touch of the eye-ball starts an action of a nerve-centre which actuates a single blink of the lids, and then the action ceases, and the lids are again opened. If the touch is intense, or is repeated, there may be several blinks in response to the irritation; but when the irritation at length ceases, the movement ceases also. Why does the movement cease on the suspension of the irritation? If there is nothing, as I submit there can be nothing, in the constitution of the centre itself to cause the arrest of its action when once it has been set going by the impact of an extraneous force, then the arresting action, like the initiating action, must come from outside. It must be imposed from without. Hence we gather that *every nerve-centre is normally subject to an inhibitory influence imposed upon it from without.*

A moment's consideration will show that this inhibitory influence is not a momentary and occasional phenomenon applied only from time to time when the cessation of activity is required. It is now an universally accepted doctrine that the whole of the nervous system is at all times the seat of a continuous circulation of currents of force of greater or less intensity. Upon every centre are continually breaking waves of force of varying intensity, some of which, taking into consideration the various degrees of instability attained by the nerve-centres, would frequently set a centre discharging at odd moments, without reference to other movements going on unadapted to any end then sought after by the organism. In short, we should all be continual sufferers from chorea. As we do not so suffer, we must therefore suppose that the application of the inhibitory influence upon a nerve-centre is not an occasional and intermittent incident in its history, but that it is continuous. That all nerve-centres are at all times subject to continuous inhibition or control, which steadies their action, prevents their discharge being initiated by inadequate impulses, and insures their return to a state of quiescence when the call to activity has ceased.

Lest this hypothesis of a universal and ever-present inhibition should appear too startling, let me hasten to explain that it is not new. The frequent use of the phrase "Loss of control" (indeed I have heard an eminent member of this society speak of "an outburst of loss of control") has surrendered the whole position. For control to be lost it must first be present; and if maintained upon some centres, why not upon all? The hypothesis already exists, therefore, and is already largely accepted; and, indeed, it is of such indispensable necessity to a comprehension of the physiology of the nervous system, that we may say of it, as the French philosopher said of the Deity: "If it did not exist it would be necessary to invent it."

We are now in a position to combine the conceptions gained of the physiological mode of action of the nervous system with the physical mode of action already arrived at, and to conclude that while every nerve-cell has its inherent tendency to discharge for ever opposed by a continuous resistance, so every nerve-centre has its inherent tendency to discharge for ever opposed by a continuous inhibition; and it is not making a very large demand upon our imagination to suppose a certain community of nature between actions so similar. In short, we may fairly suppose, that the inhibition is a strengthening of the resistance to the discharge of all the cells throughout the centre.

Thus we find that, while the intrinsic tendency of every nerve-cell and nerve-centre is ever to discharge, this tendency is continuously counteracted by an extrinsic influence which curbs it into quietude. Just as, in molecular physics, atoms are maintained in a condition of mobile equilibrium by the opposing forces of attraction and repulsion; just as, in molar physics, the planets are maintained in their orbits, that is, in mobile equilibrium, by the opposition of gravitation to their own inertia; so the nerve-centres are maintained in a condition of mobile equilibrium by the opposition of the inhibition exercised upon them to their own tendency to discharge.

Very little consideration is required to show, that to have its nervous centres arranged in a condition of mobile equilibrium is vastly more advantageous to the organism than to have them disposed in any fixed arrangement. In the first place, a mobile equilibrium is far more stable, in the sense of being less easily destroyed, than any fixed disposition. The phenomena of the gyrostat are familiar illustrations. A spinning top remains upright on its point without any external support, in spite of the inclination or the movement of its supporting surface, and in spite of deflections of its axis from the perpendicular, even of considerable magnitude. The same top, if not spinning, cannot be balanced on its point for an instant, and even, if by superhuman patience and steadiness it were to be so balanced, the slightest breath of air would at once cause it to fall.

While, on the one hand, the moving equilibrium presents a maximum of stability, on the other hand, paradoxical as it may appear, it presents a maximum of plasticity; that is to say, while it is with difficulty destroyed, it is modified and altered, within limits, with the greatest ease. Consider, for instance, the example of mobile equilibrium presented by the circulation of the earth in its orbit. So delicate is its balance that the movement of another planet, hundreds of millions, thousands of millions, of miles distant, produces a sensible perturbation in the path of our earth. It is as sensible to the presence of other bodies at these inconceivable distances as a magnetic needle is to an iron bar in its neighbourhood (another combination in mobile equilibrium). Suppose now that the earth, instead of circulating freely in mobile equilibrium, were fixed to the sun by a rigid rod. If all the other planets were massed together and brought within a mile of the earth's surface they would scarcely produce any perceptible alteration in its position in space. These examples will be sufficient to show how susceptible an arrangement in mobile equilibrium is to external influences; how readily and completely it returns to the normal after disturbance, and how difficult it is to break down. When it is remembered that the two fundamental properties of the nervous system and of nerve-centres are those of being readily disturbed from without, and of readily resuming their *status quo ante*, and when it is further borne in mind that this susceptibility to disturbance must be united with great resistance to disintegration, it will be seen how immensely advantageous it must be to the organism to have its nerve-centres in that condition of mobile equilibrium in which these antagonistic qualities are so prominent and so closely united.

Granting that every nerve-centre has its intrinsic tendency to discharge continually checked, counteracted, and controlled by an extrinsic inhibiting influence, two questions remain: What is the nature of this influence, and whence is it derived?

As to the nature of the influence, we have already considered its probable alliance with the normal resistance to the nervous discharge, but this does not carry us very far, since we are scarcely yet in a position to speculate as to the nature of this resistance. There is, however, another source from whence we may obtain a glimpse of light upon the process. This is the unquestionably undulatory character of the currents of force in the nerve fibres. Once admit, and every one does now admit, that the nerve currents are undulatory in character, and at once the whole doctrine of interference of waves is at our service to explain the phenomena of inhibition. It is more than probable that it is in this doctrine that the detailed explanation of inhibition will at length be found, and in the meantime it gives us valuable help, for just as two rays of light from the same source may be united to produce darkness, and just as two rays of sound from the same source may be united to produce silence, so we may imagine that discharges from one source may, according as they are combined, reinforce or neutralise each other.

Leaving the intimate nature of the inhibitory influence on one side, as a problem for the present insoluble, let us turn to the other problem, and ask what evidence we have of the source from whence the influence is derived. Two hypotheses are admissible. Either there may be one or more portions of gray matter specially reserved and set apart for the purpose of producing this continuous inhibition of all motor centres, thus constituting special inhibitory regions; or the function of inhibition may be carried on by the ordinary motor centres concurrently with their more generally recognised functions.

Of the existence of a single centre, whose function is inhibition and nothing else, there is no evidence, and from the nature of the case there could not but be evidence if such a centre existed. Suppose a sudden lesion, such as a blood clot, or an experimental injection, destroyed such a centre, the effect would be that the whole control would instantly be removed from every motor centre in the gray matter, and the consequence would be such a terrific spasm of the entire body as the severest epileptic fit can give us but a faint and ineffectual idea of; the spasm would at once arrest the breathing, and speedy death would surely follow. Such a malady is not clinically known, nor have all the localising experiments of Dr. Ferrier revealed the existence of any such centre. Neither is there evidence of such a centre being destroyed by a gradually invading lesion. In that case there would be excess of movement gradually increasing to universal spasm, and ending in death either by exhaustion or by arrest of breathing as before. Something corresponding to this description does actually take place in chorea, and it is at first blush conceivable that chorea might be due to the morbid weakening of a general inhibitory centre, but such an hypothesis will not bear investigation. For the movements of chorea, although affecting all parts of the body, are manifestly not due to discharge of all motor centres. Apart from their elaborate character, there is the fact that they are essentially *a succession of different movements,* and indicate therefore a succession of discharges of different centres. Whereas the weakening of our universal inhibitory centre must produce a simultaneous discharge from all centres.

The inhibition of motor centres must therefore be actuated not from a single store, but from many different places; and the next question is whether there are regions of nerve-tissue specially set apart for the origination of inhibition alone, or whether this function is exercised concurrently with others? It may be said at once that the balance of the evidence is overwhelmingly in favour of the latter hypothesis. Neither in experiments on animals nor in the experiments exhibited by disease and accident has there been any instance of a destruction of gray matter producing primarily an outburst of movement; and it is in the highest degree improbable if a number of centres existed, and by hypothesis the number must be large, whose functions were purely inhibitory, that no one of them should ever have been singled out for destruction by experiment, by accident, or by disease. Hence we are driven by a series of exclusions to the opinion, that the state of inhibition, which universally obtains throughout the motor centres, is maintained by centres which exercise this function concurrently with others.

In corroboration of this opinion there is a very large body of evidence from pathological observations. Experience at the bedside shows absolutely invariably and without any exception whatever, that whenever nervous tissue is destroyed the effect of that destruction is twofold. There is always a loss. The body or some part of the body is unable to do something that it could do before. The function of the destroyed part is abolished. In addition to this simple and direct effect of the lesion there is always, and without exception, another effect. There is always increased action of the body or of some part of it. These two results of simple destruction of tissue are called by Dr. Hughlings-Jackson the negative effect and the superpositive effect. It matters not to what pathological agency the destruction of tissue or of function is due; whether the nerve tissue is ploughed up by a blood clot, or smashed by a blow, or consumed by a morbid growth, or whether the function alone is abolished by chloroform or by ether, or by alcohol or other drugs, or whether the nerve region is merely boycotted by the severance of its communicating fibres. In any and every case in which nervous influence is withdrawn we find this double effect: the negative effect and the superpositive effect. If we cut a peripheral nerve we abolish nervous influence in the parts that nerve supplies. We find that the muscles supplied by the nerve are paralysed (negative effect). And we find that the reaction of these paralysed muscles to galvanic excitation is increased (superpositive effect). If we cut the cord, we get the negative effect of paralysis in all the parts below; and we get the superpositive effect of increased reflex action in the paralysed parts. Rupture of an artery in the corpus striatum gives rise to destruction of the tissue, and the sudden shock, with increase of pressure in the cranium, produces the apoplectic state. In the apoplectic state the whole of the voluntary muscular system is paralysed (negative effect); and the movements of breathing, the action of the heart, the secretion of the skin, are exaggerated (superpositive effect). When the apoplectic state passes off, there is hemiplegia and there is exaggeration of reflexes. When the highest nervous centres are placed out of action, either by destruction, as in general paralysis, or by suspension of function, as in alcoholic poisoning, there is an inability of the individual to do any action of high elaborateness and complexity (negative effect); and there is uproarious conduct (superpositive effect). On all levels of the nervous arrangements and in all classes of destroying or suspending lesions the rule holds absolute.

The abolition of a function is of course sufficiently accounted for by the abolition or destruction of the organ that affected that function. The negative effect of a lesion is easily accounted for. But how are we to account for an *increase* of action consequent on a *destruction* of tissue? It is manifest that no amount of destruction of tissue can of itself primarily produce increased action. In other words, the superpositive effect cannot be due to increase of action of the part destroyed. That part, so far as function is concerned, has ceased to exist. The superpositive effect must be due to increased action of some other part. Why does this other part overact when the first part is destroyed? Surely there can be but one answer; because the part destroyed exercised a controlling, inhibiting, moderating action upon the part that overacts; and the sudden outbreak of overaction is due to the sudden removal of this permanent controlling influence.

There is another circumstance with regard to the double effect of a destroying lesion which is of the utmost significance. There is always a constant relation between the negative effect and the superpositive effect. The action that is increased is always of a simpler, more fundamental, less elaborate, more general character than the action that is lost. Motion is lost, galvanic reaction is increased. Movement is lost, reflex action is increased. The voluntary muscles are paralysed, the involuntary muscles overact. The most elaborate forms of conduct are lost, the simpler forms are exaggerated. So that it appears that every nervous centre is subject to the control of a more complex and elaborate centre. In other words, the nervous centres are arranged in a hierarchy like the ranks of an army, every grade not only supplying an initiative to, but also exercising a control upon all grades below; and every grade being subject to the control and stirred by the impulse of all grades above.

There is, I submit, nothing anomalous or paradoxical in the supposition, that the same centre affords both the impulses which start and accelerate, and those which retard and arrest the action of inferior centres; for we have a daily illustration of a similar condition of things in every business organisation.

Take the case of an army, for instance. The rank and file under the orders of each corporal are at the same time subject to his control, which inhibits them from acting, and subject to his initiative which starts them into action. And as the rank and file to the corporal, so is the corporal to the sergeant, the sergeant to his immediate superior, and so on throughout the military hierarchy up to the commander-in-chief. And this is no mere fanciful or far-fetched analogy. It is a close and apposite illustration. For the whole military machinery may be actually regarded in the light of a nervous mechanism. It is by the direct action of their nerve-centres that the officers conceive and issue their orders to their subordinates, and it is by the action of their respective nervous systems that the subordinates receive these orders and carry them out. The analogy is closer still. For with orders or without orders the soldiers are continually under the influence of the military discipline, which inhibits them from taking upon themselves any independent initiative, and which provides that on the completion of any active order they shall subside into an attitude of quiescent expectancy. This continuous condition of inhibition or control is from time to time broken by behests to activity, precisely as the similar continuous inhibition of the nerve-centres is similarly broken.

Again, just as the purely local stimulus of a mote on the eyeball is responded to by the purely local action of a blink of the eyelid, actuated by a single centre of the lowest rank, so the purely local stimulus of the single footstep is responded to by the purely local action of the sentinel's challenge, delivered by an individual of the lowest rank. The analogy may be carried into much further ramifications. Though no further action follow the blink of the lid, yet the occurrence of the tickling feeling proves that the stimulus was carried beyond the local and subordinate centre up to centres far higher in rank. And though no further action follows the challenge of the sentry if it be satisfactorily answered, yet the occurrence is reported and comes to the knowledge of far higher authorities. If the grain of dust is not dislodged by the blink, but remains as a cause of irritation, further action soon follows. The stimulus is irradiated from the local centre to centres higher and higher in rank, and is now intense enough to set these higher centres in action; so that the hands are brought to the help of the lids, and if they do not suffice to get rid of the intruder, the voice requests help, and the whole body moves towards the source of relief. Similarly, if the sentry's challenge does not suffice to arrest the intruder, and his attitude is menacing and indicative of support behind, the alarm given by the sentry to his superiors is followed by action on their part. The guard turns out, the regiment is aroused, supports are sent for, the whole army may be set in motion, and a general engagement may ensue.

The parallel may be pushed further yet. A minute mote falling on a normal eye causes a single blink, and the intruder is removed, the action ceases. But the same mote falling on an inflamed eye causes a much more lively stimulus and a much more vigorous reaction. The sensation instead of being insignificant is acutely painful. The single blink is replaced by copious action of lids, and hands, and effusion of tears. As the inflamed eye to the normal eye, so is the army in the field to the army in time of peace. In the latter case the intrusion of a single stranger produces no reaction beyond what is necessary to keep him out, but the same occurrence in the face of the enemy causes a general commotion. No longer confined to the guard-room, the intelligence of the intrusion is conveyed to headquarters, and a general commotion results from the arrest of the spy.

The similarities are far from being yet exhausted. The private soldier has nothing to do with the supplies save only to take to himself the rations and clothing that are served out to him. Each subordinate officer is responsible for the condition of the clothing and accoutrements of those under his command. The colonel of the regiment, from whom proceed both the initiative to action for the whole regiment and the discipline which maintains the whole regiment ready to act, is further responsible for the supply of food, clothing, and arms for the whole of his regiment. The same combination of functions that the colonel fulfils towards his regiment the general fulfils for his brigade, the superior general for his division, and the commander-in-chief for the whole army. It is the same with the nerve-centres. Each of the lowest centres of all not only effects a movement, but at the same time governs the nutrition of the parts that affect that movement, so that when a motor nerve is cut, the muscle wastes. Each higher centre effects not only the control and the initiation of all centres beneath it, but their nutrition as well, and not only their nutrition, but also the nutrition of the mechanisms that they serve. So that while the lowest centres control the nutrition of (say) muscles only, the higher control the nutrition of the lower centres; and thereby, in a more general way, the nutrition of the muscles that the lower centres actuate, and thus control, moreover, the nutrition of bones, ligaments, &c., that are subsidiary to the movements that they directly affect. And as the commissariat is under the orders of the commander-in-chief, who governs through it the supply of nutriment to the whole army in general though not in detail, so the highest centres regulate generally, but not in detail, the nutrition of the entire organism.

If the nervous system be constituted as is here supposed, then the parallel that is now suggested will hold good not only in health but in disease. Let us consider the analogous cases and see if it do so apply.

If the sentinel is killed, he can no longer issue his challenge to the intruder, nor communicate the alarm to headquarters. And if the reflex centre is destroyed, if, for instance, the frog is pithed, the usual stimulus provokes no reflex action, and no sensation corresponding to the stimulus is felt. On the other hand, suppose the sentry is excited by drink. He is met by the patrol, and in an excess of zeal he challenges and straightway bayonets his comrade. Similarly, if the frog be strychnised, a slight and ordinary stimulus produces an excessive and extraordinary reaction.

Suppose that it is the sergeant that is killed, which may be considered equivalent to having the cord bruised. The first consequence is that the rank and file receive no orders from their superiors, the sergeant who was the transmitter of the orders being killed. Consequently they remain inactive, but they are capable of again taking part in the military operations if the messages are sent by another route, or a new sergeant appointed and the communication restored. Similarly, when the cord is cut, the centres below are cut off from communication with the centres above, and no voluntary movement becomes possible. But the inferior centres are themselves intact, and are capable of resuming their part in the bodily operations if the cord recovers, and communication is restored. There will be a second consequence of the loss of the sergeant in the relaxation of discipline which he was instrumental in maintaining. His control being removed, the soldiers will be apt to get uproarious and exceed their duty. And when the control of the superior centres is removed by bruising of the cord the reflex centres overact, and reflex action becomes excessive. Further, as the sergeant is no longer present at the daily distribution of food, the interests of his men will probably suffer, and the amount served out to those who have been under his charge will be diminished. Similarly, when the cord is bruised and the legs are paralysed, their nutrition begins immediately to suffer. At the end of twenty-four hours it is apparent that the muscles are flabbier, the skin looser and sweatier than normal.

Now let us suppose that the colonel is shot at the head of his regiment. For a short time the regiment is demoralised. It is thrown into disorder and begins to retreat. In a short time these effects pass off, and then it is found that the state of affairs is as follows: The severance of the chief channel of communication between the regiment and the commander-in-chief will greatly impair the value of the regiment to the army at large. The daily routine of the regiment will not be much affected. That is carried out by the subordinate regimental officers—by the majors, captains, &c.—and will be carried on in much the same way whether the colonel is present or no. The daily drills will be performed, the parades held, the canteen will be open, and the messes served as usual. The general knowledge of regimental duties attained by the inferior officer will suffice to carry the regiment through the simpler duties that are required of it on the march. But when there is a prospect of an engagement, when very special and precise and important and perhaps novel movements have to be carried out in the face of the enemy, then the loss of the guiding spirit is felt. When the skilful handling of the regiment, with reference to the particular nature of the ground and the presence and strength of the enemy, becomes of the first importance, then the want of a single, firm, decisive, experienced authority becomes patent. Compare these facts with the facts of paralysis from a small clot in the corpus striatum. Such a clot produces first a general shock with total paralysis of, say, the right arm. This corresponds with the initial retreat of the regiment and its virtual paralysis. When the effects of shock pass off, it is found that the arm is much weakened, and that it is weakened in such a way that the digits are altogether paralysed, the hand less so, and that the power of movement increases as we leave the periphery, while at the shoulder no defect of movements can be found. In other words, what are lost are the most peripheral movements; that is to say, the movements that are most precise, most elaborate, most special, most important, and most novel; while the movements that are least affected are the most general, the most automatic, the simplest, and the most frequently repeated. The parallel holds good in all respects, and it may be carried further. For loss of the colonel involves the omission to send in requisitions for clothing and stores. The daily rations are distributed as usual, and the regiment does not suffer at once, but as the clothing and stores become worn out they are not replaced, and thus, after a time, the condition of the regiment suffers. So with the hemiplegic limb. At first little change in it is apparent, but after a time it wastes. The loss of the colonel will not have a very apparent effect on the discipline of the regiment, for that will be maintained in a general sense by the other regimental officers, nevertheless, it must be to some slight extent impaired; and correspondingly, while in hemiplegia, we do not find reflex actions very much increased, we do find the muscular and tendon reflexes slightly increased.

Suppose now, that in the excitement of battle the colonel loses his head. He begins to send out messages to his skirmishers faster and faster. No sooner is one order sent than another follows to contradict it, another and another and another follow as fast as the messengers can succeed one another. The consequence will be that the soldiers will run first to execute one order, then to execute the next, until, as different orders come, one after another, faster than they can be carried out, the soldiers will be raised to a state of high excitement; and after a good deal of aimless running to and fro, they will at length stand still and do nothing, and finally cease acting from exhaustion. The occurrence is parallel to a fit of Jacksonian epilepsy, which begins with the hand trying, as it were, to execute many movements at once, goes on to moveless rigidity, and ends in motionless exhaustion.

We will now transfer our observations to a higher level, and consider the result of the loss of the commander-in-chief. In actual life no doubt the loss would be supplied by sending out another commander-in-chief from home, but we must suppose not only the individual lost but the function suppressed. What will be the result? The result will be that while the army maintains its general organisation intact, and while all the routine duties of regiment, brigade and division, of horse, foot and artillery are carried on much as usual, yet for campaigning purposes the army is useless. It ceases to be an assistance to its allies, a defence to its country, or a menace to its enemy. A civilian passing through its ranks and seeing everywhere the evidence of order and discipline, the trenches manned, and the sentries posted, would see no evidence of any important defect in the army. But although no defect might be seen by the civilian, the practised eye of a military observer would see that something was wrong. He would observe a languor, a want of initiative, a failure to take advantage of opportunities. Though the army might be marching in the right direction, its several divisions, perfect in their internal discipline, would not be so handled as to support one another at proper times and places. In case of an engagement, the whole army takes part, led by its generals of division, the soldiers fight gallantly, the guns are well served, the charges brilliantly delivered, one whole wing may be successful in driving back the enemy. But in the absence of a single guiding hand the result must be disastrous. The tactics may be admirable, but the strategy must fail. If there occur no general engagement, it may be long before the army is destroyed, but in the absence of a definite aim and plan, the inferior commanders become discouraged and lose their interest; the discouragement and lack of interest spread rapidly downwards from rank to rank until the whole army is disheartened. Then ensues desertion, cutting off of stragglers, failure of supplies; and soon dysentery sets in, and sooner or later the army melts away.

Corresponding to the commander-in-chief are the highest of all nervous centres—those centres whose function it is to actuate the movements and regulate the nutritions of the body as a whole, and whose activity is accompanied by consciousness. When these centres, and these alone, have their functions abolished, as in the early stages of drunkenness, and of other forms of coma, what do we find?

To the superficial observer and the layman, who are comparable with the civilian in the example, the difference is not great. As in the one case the local and routine duties are efficiently performed, so in the other are the local and vegetative functions. As in the one case no part of the army is perceptibly defective, so in the other no part of the body is conspicuously paralysed. The languor, the want of initiative, the failure to take advantage of opportunities on the part of the army is paralleled by the heaviness, the dulness, the defect of intelligence on the part of the individual. It may happen that the divisional commanders of the army are actuated by mutual jealousies and suspicions, and that when the controlling hand of the superior is removed they fall out, quarrel, attack one another, and diffuse throughout their several divisions the excitement which they themselves display. Or it may happen that, their ardent spirits no longer held in check by the wisdom and authority of their chief, they launch out into reckless enterprises, in which their troops become speedily exhausted or meet with disaster. Similarly, when the highest nervous centres are thrown out of gear, the most prominent symptoms of the loss may be an outbreak of violence or a clamorous exaltation, and this is due, it may be contended, to the uncontrolled action of centres immediately subordinate to those whose function has been lost. Such occurrences are common in the early stages of drunkenness, of insanity, and of other forms of coma.

But whether the immediate effect of the loss of the highest authority is an outbreak of excessive but misdirected vigour, or whether it is a general condition of diminished activity, in either case the ultimate result is a loss of efficiency on behalf of the body at large. We have seen how in the army the routine of discipline may be retained and the routine operations carried on, and that in the body the vegetative functions are preserved, but in both cases the highest functions which are necessary for the conservation of the organism as a whole, are lost. The march of the army is continued, but when it arrives on the field of battle the want of a coherent plan of operations loses the day. Similarly the individual in an early stage of drunkenness is competent to get to his office, but when he is there he is unable to transact business efficiently. The army without a chief becomes a *quantité négligeable* both to friends and foes; and similarly the man who is indulging in a drunken debauch is useless. Although no one part of the army is actually disorganised, and no one limb of the body is paralysed, yet throughout the whole of each body there is a languor, an inability for sustained exertion, an impaired efficiency, which, considered from the present point of view, may be regarded as a paresis of the entire organism. It is not conspicuous in any one member, because, all being equally affected, the emphasis of contrast is lacking; but if we compare the organism as a whole in its present condition with itself in its former condition, or with other similar organisms that are unaffected, it becomes at once apparent that there is a universal defect of efficiency, which in both organisations may be ascribed to the withdrawal of the stimulus of frequent positive impulses to different and new exertion—a withdrawal which is followed in both organisations by disheartening, discouragement, and diminished inclination to exertion.

The lack of initiative, the feebleness, and the possible uproar from absence of the controlling authority will be the most conspicuous, but they will not be the only effects of the removal of the highest functions either in the one organisation or in the other. In the army the supply of arms, ammunition, accoutrements, clothing, stores, and food depends on the requisitions sent home to the War Office by the commander-in-chief. If the commander-in-chief ceases to exist the stores will still be sent out, no doubt, but they will be sent in wrong proportions, haphazard and by guesswork—too much of this and too little of that. Arrived at the army, the commissariat officers sharing in the general inefficiency and carelessness, the distribution will be similarly faulty. Here will be carbine ammunition served out to infantry regiments, there will be cavalry soldiers with shoes instead of boots; in another place, horse-shoes provided for a camel regiment. The distribution of food too will be badly done. The consequence will be that the army will suffer, not only in efficiency, but in physique. The men, being badly shod, badly clad, and badly fed, will soon deteriorate in health. In the individual, too, loss of the highest function invariably causes mal-nutrition. In drunkenness and in insanity the skin becomes altered in tinge, becomes unduly dry or unduly moist; the hair is harsh and staring, or less often dripping with moisture, the nails are affected, the bowels are constipated, the breathing, the pulse, the urine are altered. To sum up, the three main consequences of loss of the commander of an army are lack of initiative, lack of control, and modification with impairment of supplies. And the three main consequences of loss of the highest nerve-centres are lack of initiative, exaggerated action, which is a consequence of lack of control, and modification with impairment of nutrition. Here, therefore, the parallel between the military organisation and the organisation of the nerve-centres still holds good. It still remains to compare the opposite error, the fault of over-action, and see if the same holds good.

Suppose the commander-in-chief to become highly excited, and to issue orders in great excess. The result will be an excess of activity on the part of the army at large, but will vary with the degree to which the orders are excessive, and with the directions in which they are issued. A multitude of contrary orders following one another faster than they can be executed will have the same effect when issued by the general to the whole army as when issued by the colonel to his regiment. They will produce a great excess of activity culminating in a standstill of consternation. The various movements will interfere with one another, so that while every man is put to far more exertion than any one movement requires, the practical result will be *nil*. The analogous process in the individual is a universal convulsion.

To recapitulate.

The propositions that I beg to submit for the consideration of the society are these:—

1. Every nervous discharge takes place normally in the face of a continuous resistance.
2. Every nervous centre is normally subject to a continuous control or inhibition.
3. This inhibition is not exercised by any separate portion of gray matter, but concurrently with other functions.
4. Every destruction of nerve tissue produces a double effect: a negative and a superpositive.

5. The superpositive effect can be produced only by the destruction of an inhibition.

6. *Ergo*, every nerve-centre not only is subject to, but exercises, an inhibitory influence.

7. Every nerve-centre controls simpler centres, and is controlled by more complex centres than itself; so that—

8. The nerve-centres are arranged in a hierarchy.

DISCUSSION.

DR. HUGHLINGS-JACKSON.

After remarking on the great value of Dr. Mercier's paper in many important ways, Dr. Hughlings-Jackson said:—

I quite agree with Dr. Mercier in the principle of a hierarchy of nervous centres. I have suggested a division of the central nervous system into three levels. (See paper, 'Journal of Mental Science,' April 1887, section 5.) The relations of these centres to one another is a very complex one. Besides relation of direction and control, the higher their evolution the more independent do centres become in their activity; there is quasi-detachment, so that in the most "detached" (the highest) centres activities can go on (internal evolution) largely independent of present converse with the environment; on the psychical side there can arise trains of thought independent of present experiences (I have considered this aspect of the subject in the paper above referred to, 'Journal of Mental Science,' section 8).[1]

I think it would be best to use the term "force" as a name for the rate of liberation of energy by nervous elements (nervous discharges). It seems to me that rate of liberations of energy by nerve cells, or as I shall say, rate of discharges, is a thing of great importance for our consideration with regard to the interpretation of some of the phenomena of epileptiform and epileptic seizures, and of their after-conditions. The bearing is particularly on ranges of convulsion, on sequence of convulsion, and ultimately on the interpretation of certain negative and positive symptoms found when the fits are over (after-conditions). It is with regard to the nature of these after-conditions that we are mainly concerned with inhibition; we are concerned at the same time, in principle, with one of the factors in all cases of insanity. I remark first on the fits.

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[1] See Spencer, 'Princ. of Psych.,' vol. i., chap. 6, footnote, and especially Section 264.

I take for illustration the comparatively simple epileptiform seizures, dealing with that variety in which the initial convulsion is of the thumb. In such a case there is a very local hyper-physiological[1] state of some nerve-cells of a part of the Rolandic region (so-called motor region)—a local "discharging lesion," or, as I sometimes call it, a "physiological fulminate." It is well known that there are many ranges of convulsion in such a case, from that seemingly limited to the thumb, to universal convulsion. With regard to ranges we have not only to consider the number of cells highly over-unstable in the local "discharging lesion," but their degree of instability—not only the different quantities of "explosive" matter making up physiological fulminate, but the different degrees of "explosiveness" of that matter. Supposing a quantity of gunpowder and a quantity of gun-cotton (each having the same quantity of energy), the effects of the explosion of the two would be very different, the latter exploding so very much more rapidly. I submit that *one*[2] factor governing range of convulsion is rate of the discharge; I speak now of the primary discharge, that is, of the discharge of the cells of the "discharging lesions."

We shall speak of rapid and slow discharges in epileptiform seizures. All discharges producing paroxysms of convulsions are very rapid in comparison with the discharges in health. But the abnormally rapid discharges producing convulsion differ in rate; they are comparatively with one, another, rapid and slow. I fear "rapid" and "slow" are not the right words. I do not mean that "nerve impulses," after discharges which produce convulsion, "travel" faster than those after the discharges of health; but that in the former case more numerous "nerve-impulses" are emitted in a given time, or else that more cells are simultaneously discharging upon the same part, "a greater quantity of motion" in a given time. (The reverse for "slow discharges.") If the primary discharge in a fit be slow, slow in the sense I mean, the "currents developed will "flow" more exclusively to that part which is most specially represented by the cells of the nervous arrangements of the "discharging lesion;" in the case chosen for illustration, say, to the thumb only. If the primary discharge be rapid (the same quantity of energy being liberated as in the former case, that of slow discharge), the currents will "flow" also to parts less specially represented by cells of those nervous arrangements, not only to the thumb, but to other digits also; the convulsion will be of greater range. To say that a part of the body is most specially represented in a centre, means that it is united by lines of least resistance to that centre, the parts less specially represented in it being united to it by lines of greater resistance. Let us arbitrarily suppose three degrees of resistance between the discharging lesion and three parts of a muscular region it represents; (1) least, (2) more, (3) most. A very slow discharge may be limited to (1), a more rapid to (1) and (2), and a most rapid will involve all three parts. We are supposing that the same quantity of energy is liberated in the three instances, urging that the more rapid liberations overcome greater resistances, and thus that there is wider spreading of effects on the periphery. The more rapid the discharge, the more nearly simultaneous will the convulsion of the three parts be; the less obvious the sequence, the more nearly equal will be the degree of convulsion of each, and the sooner over will be the attack; speaking very roughly indeed, the effects of a more rapid discharge will be spread out more widely, more thinly, and will last for a shorter time. The slower the discharge, the more of a decided sequence will be the convulsion, the less equally will be the degrees of convulsion of each part and the more lengthy the attack; the part first involved will suffer most. No doubt in all cases there is a *compound sequence*, but in very rapid convulsion we may not trace it; in slow convulsion the compound sequence may be traced and symbolised as x, then $x^2 + y$, and then $x^3 + y^2 + z$.[1]

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[1] The pathological processes (disorder of nutritive process) leading to the local hyper-physiological state does not concern us here.

[2] I say one factor, because we have to consider the number of cells highly over-unstable as well as degrees of high over-instability of them. Further, in paroxysms in cases of epilepsy proper, the "discharging lesion" (highest centres) will be of cells of nervous arrangements which are highly integrated, that is, nervous arrangements (or units) which represent great ranges of the body in very intricate combinations. In the case of epileptiform seizures (middle motor centres) they will be all of less integrated nervous arrangements. The discharge of the higher centres will, on account of the higher degree of evolution (factor integration) of those centres, produce wider and more simultaneous effects.

I have so far spoken as if the discharge were primary only, that is, as if it were of cells of the "discharging lesion" only; possibly it may be so in very slightest local seizures, as when the spasm is of the thumb and scarcely at all of other parts near to it also. But when a convulsion starting in the thumbs spreads greatly in range (say to the arm, face and leg, or further still), the supposition is that the primary discharge, that of the fulminate (otherwise "discharging lesion"), upsets the equilibrium of, that is discharges, *normal* cells, with which the cells of the fulminate are connected, and compels them to "co-operate in its excess"; there are primary and induced secondary discharges. But it is supposed that the rate of the primary discharge always governs the situation—that rapid discharges lead to wider-spread, to more equal convulsion of the parts engaged, to more temporary convulsion and to convulsion of less distinct sequence; whilst slow discharges lead to more local, more unequal convulsions of longer duration and more decided, more traceable sequence. (The same quantity of energy is supposed to be primarily liberated both in the rapid and slow discharges (by the "discharging lesion").)

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[1] Nothing beyond rudest quantification is of course meant by these symbolisations, in spite of the employment of definite indices. I have often urged that we should pay attention to compound order of increase of paralysis, dependent on increasing gravity of destructive lesions, and to compound order of spreading of convulsion dependent on increasing discharges. Nervous evolution does not imply insensible gradations, but increases in compound order. I suppose that increase in compound order, as roughly symbolised in the text, applies to all increasing activities of the nervous system (including those correlative with mental states) both in health and disease.

I now consider post-paroxysmal states. After epileptiform seizures there is paralysis. I submit that after slow discharges (slight fits long continued and limited in range), the paralysis is very local and decided, and therefore obvious; after rapid discharges (severer shorter fits) it is wide-spread, "diffuse," and therefore easily ignored. In the former case it lasts longer than in the latter. I submit that in the former case there is really less paralysis than in the latter. This, however, is denied: the very contrary is affirmed. As an objection against an hypothesis I contend for (essentially Todd and Robertson's). The assertion has been made that there is more paralysis after slight than after severe fits. It is said that whilst after slight epileptiform seizures there is decided paralysis, say, of one arm, there is none after the severest epileptic seizures; my contention is that the whole physical condition in the latter is paralytic, and that there is very much more paralysis than in the former case.

Applying Todd and Robertson's hypothesis to post-epileptiform states, and bearing in mind what has been said as to the difference in the effects of slow and rapid primary discharges (of the "discharging lesion"), and consequently in the secondary discharges (of normal elements), I submit that after slow discharges the post-epileptiform exhaustion of central nervous elements is limited in extent, very local, very decided in degree, and that it remains for a comparatively long time; whilst after rapid discharges the exhaustion is wide, diffuse, slight in degree, and remains for a short time.

I now apply this generalisation to after-conditions of paroxysms of epilepsy proper, and thus come closer to the object of Dr. Mercier's address. We have here to do with the highest cerebral centres—popularly "mental centres"—epileptic paroxysms being owing to "discharging lesions" of small parts of these centres. These after-conditions are, scientifically regarded, cases of insanity; what is called epileptic mania is one example. Epileptic mania should be called "post-epileptic 'loss' of consciousness" (a purely negative mental state), with "mania," (a diametrically opposite, a super-positive mental state). Post-epileptic coma is certainly an insanity; it is acute temporary dementia (see Dr. Mercier's admirable contribution to the realistic study of insanity, a paper entitled *Coma* ('BRAIN,' January 1887, especially p. 483). In all other cases of insanity, the condition is essentially similarly duplex; there is in every one some degree of negative affection of consciousness, and in every one there are also some super-positive or positive mental manifestations, except in absolute dementia, if there be such a thing, when the mental condition is entirely negative. Of course our proper concern, as physicians, is not with negative affections of consciousness, but with the physical states they imply. I submit that, taking the case of epileptic mania, the "loss" of consciousness answers to what is physically exhaustion (functional absence) of elements of the highest "layers" of the highest centres (Dissolution), and that the mania answers to activity of the next lower "layers" and rest of the nervous system (the mania signifies the Evolution going on in what is left intact of a maimed nervous system). But the further supposition is that, the higher "layers" of the highest centres being functionally absent, the next lower layers are uninhibited, and are in consequence in greater activity than normal. Before saying more on this point I would clear the ground by stating what I think are the four factors in post-epileptic and in all other cases of insanity.

The factors are: (1) Different depths of Dissolution of the highest centres and consequently different heights of Evolution remaining.[1] (2) The rate with which the Dissolution is affected. (3) The nervous system in which the Dissolution occurs (the person, young or old, clever intelligent, timid or brave, &c. &c.). (4) The influence of external conditions and of local bodily states.[2] I shall consider only the second factor.

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[1] If we distinguish between uniform and local Dissolutions of the highest centres, there would be five factors in cases of insanity.

[2] I have been misunderstood to have put forward the hypothesis of Evolution as a basis for the clinical classification of insanity. For clinical purposes we must have arrangements of them by type, for a long time to come at any rate.

I submit that the more rapidly Dissolution is effected in cases of insanity (the more rapidly the higher layers of the highest centres lose function), and thus the more quickly control is removed (from lower "layers"), the greater is the rate of activity on the lower level of Evolution remaining, and correspondingly the more super-positive are the positive mental symptoms. If we take extreme cases, we see that the senile dement whose Dissolution is effected with very great slowness is quiet; whereas the post-epileptic maniac, who has undergone Dissolution some thousand times more rapidly, is the most furious of all maniacs (activity at a very great rate is what here concerns us). I wish to show the bearings of what was said on different rates of discharges in epileptic fits, and on the varying condition of the highest centres after them, on the symptoms of post-epileptic insanity. (I am still neglecting three out of the four factors in insanity.)

In all cases of post-epileptic insanity Dissolution has been rapidly effected, but there are degrees here; in some it is comparatively slowly, in some comparatively rapidly effected. Clinically there are after some very slight epileptic fits during "unconsciousness" exceedingly elaborate[1] actions, which to bystanders may seem nearly, if not quite normal. These actions are also quiet (at no great rate), and are long continued. The post-epileptic condition has, in these instances, the three characters (i.) great complexity, &c., of actions at but (ii.) little rate (busy, perhaps, but not rapid), and (iii.) lengthiness. I submit that the discharge in the prior fit was slow, and consequently that the exhaustion in the highest centres effected by it, remaining when that discharge had ceased (Dissolution), is very limited in extent, very local, very slowly effected, and very decided in degree; consequently the lower level of evolution remaining in those centres is high, the activity on that level is at a slow rate and is long continued. Supposing that there are "all degrees" (which here I neither deny nor affirm) from such cases to cases of furious post-epileptic unconsciousness with mania, I will take the last cases in further illustration. I submit that in these the exhaustion in the highest centres after the fits (Dissolution) is very wide, "diffuse," was rapidly effected and is very slight in degree,[2] and that the level of Evolution is low, that the activity on it is at a great rate and of short duration.

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[1] We have nothing to do here with the "importance" of the actions after epileptic fits, but with their degrees of complexity, speciality, &c.

[2] Not being an Alienist physician, my experience of these degrees of post-epileptic states is small. I deal with cases I see, and speak subject to correction by those whose experience of what are called mental diseases is greater than mine. I by no means pretend to explain the various ways in which temporary insanity is associated with epileptic fits.

I have nothing to say on the nature of the inhibitory process. I would urge, however, that here, as in all other cases, we should distinguish between the psychical and the physical; we cannot legitimately speak of any mental states as inhibitory of any physical processes, any more than we can speak of them as producing movements. I have long urged that controlling as well as directive action is displayed throughout the nervous system.[1]

It is quite certain that we have in some way super-positive effects consequent on negative lesions; indirectly consequent, of course. I have illustrated this dogma sufficiently in one way in the preceding remarks, and have considered it with regard to the temporary insanity produced by alcohol and the insanity of starvation in the "Croonian Lectures" ('Brit. Med. Journal,' April 12th 1884). The dogma is, I think, applicable in cases where questions of inhibition do not arise. "Disorders of co-ordination" in cases of negative lesions are, I submit, cases of paralysis; the negative lesion, that caused by the pathological process, causes paralysis only, paralysis in the sense of loss of some movements of the regions affected (the most special); what is called the disorder of co-ordination is the "forcing" of (over-development of) other movements of the same region (the more general) by activity of healthy nervous elements, in the fruitless endeavour to develop those no longer possible. If, in the case of the ataxy of tabes, the negative central lesion does not cause loss of movements by destroying any motor nervous arrangements, there is nevertheless (obviously in an early stage of this "disorder of co-ordination,") underdoing of some movements and overdoing of some others. In an early stage we see defect in the movement in which the peroneus longus is chiefly concerned, and overdoing of that in which the tibialis anticus is chiefly concerned. (I have considered this matter in a paper, "Journal of Mental Science," April 1887, Section 10.)

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[1] The following is from my second "Croonian Lecture," 'Brit. Med. Journ.,' April 5, 1884: "Roughly, we say, that there is a gradual 'adding on' of the more and more special, a continual adding on of new organisations. But this 'adding on' is at the same time a 'keeping down.' The higher nervous arrangements evolved out of the lower keep down those lower, just as a government evolved out of a nation controls as well as directs that nation. If this be the process of Evolution, then the reverse process of Dissolution is not only 'a taking off' of the higher, but is at the very same time a 'letting go' of the lower. If the governing body of this country were destroyed suddenly, we should have two causes for lamentation; (1) The loss of services of eminent men: and (2) the anarchy of the now uncontrolled people. The loss of the governing body answers to the Dissolution in our patient (the exhaustion of the highest two 'layers' of his highest centres); the anarchy answers to the no longer controlled activity of the next lower level of evolution (third 'layer')."

Dr. Francis Warner.

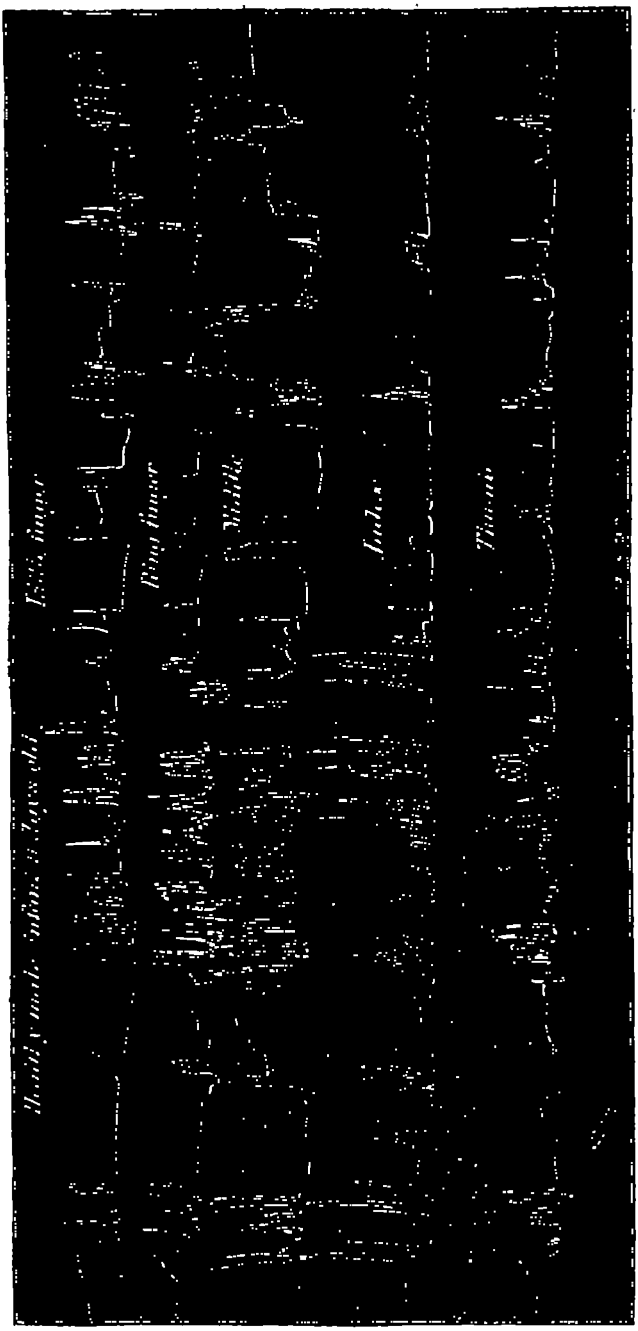
The study of Inhibition is so wide a subject, that I propose to confine my remarks to cases where motor action of the brain stops sequent to an impression received through the ear and the eye. It is proposed to consider observations as to what may be seen sequent to the period of inhibition; together with inferences as to what occurs during the period of no outward display of action in the brain.

Tracing 1 (see Fig., p. 394) shows spontaneous movements of an infant during fifteen minutes; the method employed is described in the 'Journal of Physiology,' vol. iv. No. 2.

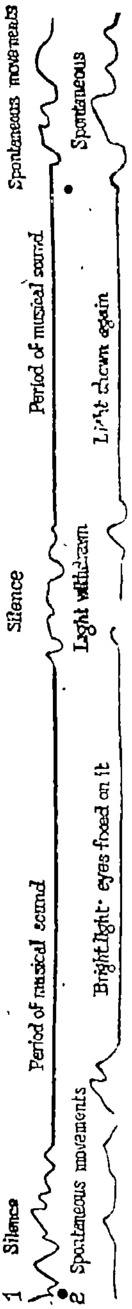
Tracing 2 shows the spontaneous movement of an infant five months old; this is seen to be inhibited by the sound of a musical-box, or by a strong light. The effect of the sound, or the light, was to arrest or inhibit movement temporarily; our chief concern is to study what occurs in the brain-centres during the time of no movement, *i.e.* during the time when no efferent currents are passing from the centres to the muscles.

I will not stop here to argue that light or sound, acting upon the eye or ear respectively, do produce a physical impression on the nerve-centres. I have elsewhere enunciated the proposition that "every vital act requires among its antecedents a supply of pabulum and stimulation by some force;" having previously given evidence in support of that proposition, I will not repeat it here. I have also shown that an impression produced upon a nerve-centre or group of centres may be delayed in its expression, and known to us only by subsequent series of movements. I now proceed to suggest how we may observe the results of what occurred in the centres during the quiet time when no efferent currents were passing from it. Common experience shows that after exercising the child frequently, say twice a day for fifty days, "he has learnt to know the musical-box," and this is indicated by the exact repetition of a particular series of movements, or efferent currents proceeding from certain nerve-centres to the muscles following the sight or sound of the box. A getting ready for action among the centres resulted during the one hundred periods of inhibition.

Now for a second example. Observing a school during a drilling lesson, I saw the first, second, and third classes go through the same exercises to the same words of command. The first class had



TRACING 1.



TRACING 2.

been similarly drilled, say, one hundred times, the second class sixty times, the third class only twenty times. In the first class the exercises followed in their detailed series of nerve-muscular movements with great uniformity, exact in the time and quantity of movement in each part, and each group of nerve-centres. The command is followed quickly and surely, by the particular series of movements. The exercise has been well learnt by this first class; it is less exact in the second class. The third class did not "know" the exercises, they (*i.e.* their nerve-centres) had not been taught sufficiently; they must be further impressed through eye and ear before their exercises become as exact as those of the first class. The children being required to stand in order motionless, and unimpressed by other sights than the teacher, they see the exercises as he performs them (sight impressions), then at the word of command (sound impressions) they (*i.e.* these centres) perform the same or similar series of movements.[1]

Returning to what took place in the baby's brain, after one hundred impressions by the light, and the sound of the musical-box, we find his centres tending to act in groups, as indicated by definite groups and series of movements. It may be inferred that some kind of change has been produced among the nerve-centres, forming them into functional groups, or groups functionally connected for action. Some explanation of the hypothesis of functional unions of centres must now be given. When a definite combination of movements of certain parts (*i.e.* combination of centres sending efferent currents) follows with uniform exactness upon a certain stimulus, it may be assumed as a working hypothesis that those centres have in some way—I do not know how—become "tied," or functionally united for action. If a stimulus, N, is uniformly followed by movement of the parts A, B, C, D, it is assumed that the centres a, b, c, d, coresponding, are in some way tied, or compelled to act together. It would commonly be said, the more uniform the recurrence of the action the more automatic it is. During the period of inhibition the centre is being stimulated its nutrition is probably as high as at other times, but no efferent currents are passing from it to the muscles, there is no expenditure of force in that way, the nerve-force is being spent in making ties or special unions. Observation of what follows shows us what has been occurring during inhibition; functional unions have been formed and the centre got ready for combined action—work was being done during the quiescent stage. Observation of the delayed expression of the impression produced during inhibition, affords evidence that functional ties or unions of centres were formed.

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[1] See 'Anatomy of Movements,' p. 77.

In the third class at the drilling-lesson, unions among nerve-centres are formed, while the children are quiet and looking at the teacher; the efferent expression of those unions is seen in subsequent movement on the command being given.

In this union of nerve-centres, while sending no efferent currents to muscles, I believe that we have the representation of some forms of brain action concerned in producing thoughts, or rather the brain state corresponding thereto. There seems to me no evidence that the period of inhibition in the cases given is a period of no action in the nerve-centres, but rather that it corresponds to the molecular changes which represent the mental state called "attention," or "getting ready for combined action among nerve-cells," and the physical process of "learning." All expression of mind in action is by movement, and the expression "unions of cells" may be delayed indefinitely, not appearing till some fresh stimulus, such as the word of command or some object seen, stimulates efferent currents from such groups of cells expressed by series of movements.

Dr. W. H. Broadbent.

I think Dr. Mercier will find the Society prepared to go with him a long way in the direction of the idea of Inhibition which he has brought before it. For my own part, I have never accepted the hypothesis of special inhibitory centres, but have looked upon inhibition as a simple corollary of the action of one nerve-centre upon another—command implies control. It has, indeed, seemed to me that the mystery surrounding the question of inhibitory influence, of which Dr. Mercier speaks, is very much of our own making, and is a mystification rather than a mystery.

The simile of an army, which he employs, is one which must have occurred to many minds, and I have some recollection of its use before as an illustration of the subordination of lower to higher centres in the nervous system; but he makes a new and ingenious development of it, in order to get rid of the necessity for special trophic centres and nerves, distinct from other centres and nerves, in which conclusion I agree with him, although it would be easy from the same comparison to argue the reverse.

I cannot, however, accept the idea, on which so much of Dr. Mercier's reasoning turns, that there is any necessary inherent tendency of a nerve-centre which has begun to discharge to go on discharging until it is utterly exhausted. There is just as much reason to assume, that a muscle once called into action must continue to contract till its store of energy is expended, for, like a nerve cell, it is a reservoir of force. All nervous action is in response to some stimulus, and it lasts only so long as the stimulus which calls it forth continues. When light falls upon the eye, or sound upon the ear, a series of nerve action or discharges is set up, which ceases with the cessation of the visual or auditory impression, and, in like manner, central discharges down the motor-tract are conditioned by some determining influence. A nervous discharge may be compared to the ringing of the electric bell, which begins and ends with the pressure on a button, which, by making and breaking the circuits, conditions the evolution of stored-up energy.

A minor criticism is a repetition of that already made by Dr. Hughlings-Jackson, in which he points out that the tremor of paralysis agitans and the tremor of drunkenness, or of exhaustion, are of a totally different kind, and cannot possibly illustrate the same condition. Like Dr. Jackson, I have an hypothesis as to the seat of the morbid change in paralysis agitans, which, emboldened by his example, I venture to mention; I believe the lesion to affect the muscular nerve endings. I may, perhaps, be permitted to add also, that the few epileptic fits which I have happened to see, have not ended in the terrific universal spasm which he describes, but more frequently in a series of general spasmodic jerks of diminishing intensity.

The idea as to the nature of nerve energy or nerve force, which underlies Dr. Mercier's reasoning on the subject of inhibition, and which is generally accepted, is that each cell is a reservoir in which force is stored, and each fibre a channel by which force is carried; the whole conception is one of liberation and transmission of energy. An impression on a sense organ sends a wave of force up to the centres; a motor impulse is an explosion in certain cells of the cortex, and the energy set free travels downwards till it impinges upon given muscles and calls them into action. I have for a long time held a different hypothesis of the nature of nerve force, and of the relation in which the nerve centres stand towards each other and towards the peripheral nerve-endings. This is that the different centres among themselves, and the centres and the peripheral terminals of the nerves in muscle or sentient surfaces, hold each other in mutual tension, the periphery reacting on the centres in immediate relation with it, and the lower upon the higher centres, as well as the higher upon the lower and upon the periphery, and that a nervous impulse is not so much a transmission of energy as a disturbance of equilibrium. The lower centres and motor nerves and muscles do not passively await the shock of the explosion in cortical or other cells, but are constantly in touch with these cells by a sort of back pressure. The best illustration of the mode of action which I believe to obtain throughout the nervous system, is furnished by telegraphy. When a message is sent across the Atlantic, it is not a flash of electricity which starts from the battery here, travels along the wire and affects the needle at the other side. There is a battery at each end, and the connecting wire is maintained in a condition of electrical tension or charge. What happens in the transmission of a signal is that disturbance of equilibrium at one end determines a corresponding disturbance of equilibrium at the other. It is in virtue of the susceptibility, due to this state of charge in the connecting wires, that multiple and opposite messages can be transmitted at the same time along the same wire. In the same way it is not force generated or liberated in the cells of the cortex, and transmitted to the muscles which sets them in action, but the entire chain of centres and the nerve terminals being in a state of tension or charge, energy liberated in certain cortical cells disturbs the equilibrium of corresponding lower centres, and ultimately of the muscular terminals, which last, as by the pulling of a trigger, set free the latent energy of the muscular fibres. To employ another more crude and less exact analogy, the difference between the received view of nerve-action and that which I am attempting to set forth, may be compared with the transmissions of energy by means of water dashing down an open channel and through a closed tube.

This idea of the relation between the nerve-centres and the periphery, in which, not the centres only, but the nerve-endings and the structures in which they are distributed exercise the office of batteries, which maintain the connecting nerve-fibres in a state of charge, seems to me to render more easy of comprehension various phenomena normally exhibited by the nervous system, and it may, when more fully worked out, throw light on morbid phenomena. For example, it gives to the peripheral end-organs an importance contended for by many investigators and thinkers, but generally ignored. Ganglionic powers, it is true, are conceded to the retina and to the expansion of the auditory nerve, scarcely inferior to those of the cerebral cortex, but corresponding powers must belong to the cutaneous nerve-endings. Muscle, again, has been said to be only less nervous than nerve; on the view here contended for, this follows naturally, and muscular tonus is an expansion of the tension assumed to exist.

Again, the degree of tension maintained throughout the nervous system may be high or low. When it is low, there will be diminished stability, and, as a result, the effects which Dr. Warner has described and represented graphically before the Society.

In telegraphy the equilibrium may be disturbed either in a positive or in a negative direction, and it is not impossible that some parallel to this may occur in the nervous system. It would follow, too, on the hypothesis of mutual resistance of the centres among themselves, upwards as well as downwards, on both the motor and sensory sides of the nervous system, that sudden withdrawal of this resistance on the part of a lower centre might determine discharge in a higher centre; in this way, epileptiform convulsions might have their starting-point in the basal ganglia or in the cord.

So far the illustrations are taken chiefly from the motor side of the nervous system, but, of course, the relation of tension will exist between the sensory and motor sides of the nervous system, between the sensory and motor nerve-nuclei at the same level in the cord, which are concerned in simple reflex action, between the basal sensory and motor ganglia, and between associated sensory and motor areas in the cortex; between the two hemispheres, and between the cerebrum and cerebellum, and between these again and the cord.

Inhibition is a simple corollary of the relation here supposed to exist between the different parts of the nervous system. The increased knee-jerk induced by pulling the hands is an expression of the intensified general tonus, and the inhibition of reflex and other movements by powerful impressions on sensory nerves, which finds no place in Dr. Mercier's theory, is explained by the heightened tension thereby induced, primarily in the related centres, and then throughout the nervous system.

Trophic changes seem to me to be more comprehensible when the importance of the nerve terminals is recognised, and their relations with the tissues are considered. The ultimate ramification of the nerves, in the structures to which they are distributed, are so completely lost in these structures that they may be said to form part of them, unless, indeed, we say that the structures, especially such structures as skin and muscle, are the nerve terminals, instead of saying that they receive them; they have everything in common, and whatever affects the nutrition of the one affects that of the other. On the one hand, then, the state of nutrition of the tissues will determine the degree of tension maintained in the periphery of the nervous system, and, on the other, the tension prevailing in the nerves will influence the nutritive operations in the tissues. It is interesting to note that, in the most familiar trophic derangement of nutrition, Herpes Zoster, the disturbing influence travels in the opposite direction to the normal functional impressions in the nerves affected, which are sensory and centripetal.

Another recommendation, which the view of sustained charge and mutual tension of the nerve-centres and periphery, which I have attempted to sketch, has for my mind, is that it renders somewhat less incomprehensible the extraordinary phenomena seen in hysterical women, which Charcot has brought to our knowledge. Physiology, as yet, has scarcely taken them into account, and in nervous pathology they stand apart, and are not correlated with other diseases. A distinguished friend of mine indeed once went so far as to say that woman ought to be excluded from the domain of nervous pathology. In my opinion, on the other hand, the physiology and pathology of the nervous system will have to be rewritten from the point of view opened out by these phenomena. There is no structural difference between the nervous system of a hysterical subject and that of a healthy man, and the hemi-anæsthesia with its transference from one half of the body to the other, and the hypnotic phenomena, betray tendencies and reveal potentialities and relations dormant in the healthy nervous system. Now, if all parts of the nervous system are in a condition of charge, and hold each other in mutual tension, the transference of hemi-anæsthesia becomes in a way comprehensible; and the comparison which makes hypnotism a state of polarisation of the nervous system is in some measure justifiable.

Nothing could be easier than to find objections to the view here crudely set forth. At present, however, it is simply a tentative hypothesis, and is placed as such before the Society, and it would be premature to state and discuss the difficulties which suggest themselves.

DR. SAVAGE.

I wish to ask Dr. Mercier if he makes any distinction between Control and Inhibition?

I confess that I rise with diffidence, because I do not yet fully know what this inhibition is: if it is only control, then I do know something about it; but if not, then I shall only expose my ignorance in speaking.

I can understand control and moral control, or self-control, but moral inhibition I cannot grasp as a thing apart.

As to control, it seems the whole matter is part of our very existence, we learn control in one way or another or we perish. The facts of control are the facts of developing in our environment. We are moulded or controlled by our surroundings.

Dr. Warner has pointed out that the infant is in a state of uncontrolled restlessness. Quite so; it is learning what it can and what it cannot do, and those children who never learn, become idiots or perish; they have no power of control, which comes to this, they have no power to store any energy.

At the other end of the scale I see the persons in Asylums who have lost this power of control, and as a result fall out of relationship with their surroundings.

All theories and hypotheses have to run a pretty severe fire before they are accepted, and with Dr. Mercier's there will be the same experience, for though it covers many wants it does not comply with all. I do not understand the fluid, or whatever it is, which is manufactured, stored and distributed through the nervous system, for among the insane I meet with very surprising cases, if they are to be explained on the fluid theory. Thus I remember a patient who having had a very severe series of fits affecting one half of the body, passed into post-epileptic stupor. This lasted but a short time, during which she was hemiplegic; she awoke in a state of acute mania, using all her limbs with great vigour. This stage lasted some hours, when again hemiplegia was present, and this lasted for some weeks. Several similar attacks occurred before the death of the patient, and no coarse lesion was discovered. That any theory of storage and expenditure will explain this, I doubt; but I do not think that the suggestion of Dr. Broadbent, that the communication along the nervous lines depends on tensions, is much more likely to be true, and may be made to explain this and similar cases.

I do not agree with all the facts of Dr. Mercier. Thus, when he says that if we "have tried hard to stifle a laugh or yawn, and if it breaks out in spite of the inhibition, it is more violent than if we had at first given way to it; and the same is surely true of an outbreak of temper." I can only say that this last appears to me to be a very unchristian doctrine, and would not tend to the good of society, as no one would be at the risk of checking his little tempers lest they became horrid rages. We are very much dependent on hypotheses for the development of our knowledge, and we depend greatly on our hypotheses by the use of similes. Some object to this, but it is the old and recognised plan to explain the unknown by reference to the known, and the more we study nature the more are we forced to believe that one set of laws governs every action; if we only get at the right set of laws, then we have nothing to do but fit them. The parallel given by Dr. Mercier of the hierarchy of control as seen in the army is very pretty. As far as the serial arrangement is concerned this is correct; but it will not do to push likeness too far, for we are taught that the highest nervous controlling parts are the latest developed and the least stable; whereas in the hierarchy of command in the army, surely the Commander-in-Chief is not the youngest and latest development, nor is he the least stable.

The paper is one which has made us think, and we have reason to be grateful to the author.

Dr. Cattell.

It may be worth our while to spend a few minutes in considering Inhibition from the mental or psychological side. The subjects with which the Neurological Society is concerned can often be approached both from the side of the nervous system and from the side of consciousness, and we must ignore neither the one nor the other of the two great highways leading to knowledge. It so happens that in the case of inhibition the first suggestion and first advance were made by studying the mental process. Herbart, seventy-five years ago, laid great weight on the arrest or inhibition of one idea by another, and developed mathematically an elaborate statics and dynamics of the mind. Obstructive association, it will be remembered, is a term occurring in English psychology, and recently much attention has been given to the subject by psychologists, both on the Continent and in England.

I have read Dr. Mercier's paper with great interest; but there is much in it with which I cannot agree. Analogies are in better place as illustrations than as arguments. I cannot admit Dr. Mercier's interpretation of "the first law of motion"; indeed it seems to me that he brings forward his own *reductio ad absurdum* when he says, in concluding, "(7) every nerve-centre controls simple centres, and is controlled by more complex centres than itself," thus picturing an endless series of nerve-centres always becoming more complex. It seems more reasonable to suppose that nerve-centres inhibit (using the word in its broadest sense) themselves and each other. It is indeed this fundamental property of the nerve-cell, that of storing up energy or inhibiting itself, on which all life and development depend. There could be no growth if all afferent impulses were immediately transferred into efferent; it is owing to the fact, that part of the energy coming from without is stored up in the body, that the individual grows and the race develops. Looked at from the side of the mind, we see how in the lower animals, and even in savages and children, action is chiefly determined by the immediate surroundings, and we can note the gradual growth of control or inhibition, until in the developed man, thought, feeling, and action depend on the sum of impressions which have been inherited and gathered.

Our mental life is a continuous process of association and inhibition. We can only attend to ideas of a given degree of complexity, and we can only act in one out of a large number of possible ways. Thoughts, feelings, and actions are continually helping and continually hindering each other. The train of our ideas and the succession of our movements are the resultant of these forces. We cannot think of the shield as both silver and gold; we cannot both eat our cake and keep it. We may perhaps look on desire as motive, and will as the result of the conflict and inhibition of desires. Our character depends largely on the relation between motive and inhibition. Some yield readily to appetites and emotions; others have what we call self-control. Motives may be so carefully weighed against each other that no action follows, as in the case of Hamlet, who continually looked "before and after." Some drugs, as opium and alcohol, seem to paralyse the inhibiting power of the will. The refreshment from sleep is, doubtless, largely due to the rest of the inhibitory functions.

Inhibition is a subject so full of interest to the student of psychology, that I am tempted to say more; but in order not to prolong this discussion, I shall confine myself to calling special attention to the important papers on Inhibition, by Dr. Brunton in 'Nature,' and Dr. Gaskell in the 'Journal of Physiology.'

DR. MERCIER'S REPLY.

Underlying the whole of Dr. Jackson's remarks there is the assumption, that inhibition is exercised by all nerve-centres, and that the views I have advocated are correct. It would perhaps be more accurate to say, that this agreement indicates that I have correctly set forth Dr. Jackson's views. Granting that the results of the removal of inhibition vary with the rapidity with which the inhibition is removed, yet in any case the inhibition must first be there, and this is my contention.

I should scarcely regard the examples given by Dr. Warner as instances of inhibition. In the highly nourished and actively changing tissue of the young brain the general nervous tension is normally high, and these vague undirected movements seem to me to be due to a bubbling over, as it were, of superfluous nervous energy. When a novel impression is made on the sense-organs, this energy is not so much inhibited as drafted off in new directions and utilised in other ways.

Dr. Broadbent's contribution to the debate appears to me extremely valuable. It is a great gratification to me to find that he agrees to the non-existence of special inhibitory centres, and of special trophic centres and nerves. His suggestion, that such structures as skin and muscle *are* the nerve terminals and not merely receive them, appears to me, from this point of view, absolutely correct. Considering the effect that nerve-lesions have upon nutrition, considering that the function of these organs depends on nervous transmission, and having regard to the fact that function and nutrition are two sides of the same process, it appears to me inconceivable that the constant wash of nerve-waves upon a tissue should not affect and regulate its nutrition.

With regard to his hypothesis, that the connection between the nerve-centres and the nerve-endings should be looked upon more as the disturbance of a tension than as the transmission of a force, I am unable to give to this so unqualified an adhesion. I would ask if there is not some danger of our being led astray by the difficulties of nomenclature, for after all what do we mean by a tension? Do we not mean a force that is struggling to escape? that is struggling to overcome a resistance? And is this not substantially the same state of things as I have suggested to exist in the nervous system?

In answer to Dr. Savage's question, I must confess that I am unable to draw any distinction between control and inhibition; and that the two terms, when used to connote a purely physical process, an affair of nerve-cells and molecules, may be used interchangeably. But control, so understood, has nothing whatever to do with moral control; and if it pleased any member of the Society to refer to this latter as moral inhibition, I know of no valid reason against his doing so. Dr. Savage must allow me to protest against his fathering upon me a theory, that the influence that travels in the nerves is a fluid. I speak of it always and everywhere as a force. If Dr. Savage was struck with a stick, surely he would be able to grasp the stick as a thing apart from the blow.

With regard to the hierarchical arrangement of the nerve-centres Dr. Savage warns me of the danger of pushing an illustration too far, and both he and Dr. Cattell consider, that the hierarchical hypothesis is altogether ruined by the want of any control for the highest centre of all. But the fact is that these difficulties present themselves because the parallel was not carried far enough, and the analogy was not worked out to the end. If we pursue the matter to its conclusion, we shall find that the highest centre of all is represented not by the Commander-in-chief, but by the War Minister, or rather by the Cabinet, and that the control of this highest and most authoritative body is furnished, not by any single superior authority, but by the public opinion of the nation at large. It is the controlling check of the continual streams of information as the state of public opinion in the country, as to its resources, &c., and as to the relation to the country of affairs abroad, that determines the course of the Cabinet; and it is the controlling check of the streams of nervous energy arriving from the body at large, and from the outside world through the special sense-organs, that is the inhibiting agent on the highest of all nerve-centres. Even, however, if my explanation of inhibition should not be correct, I submit that the hypothesis of a series of nerve-centres, each controlling those below and being controlled by those above, is at any rate simpler and more comprehensible than Dr. Cattell's notion that nerve-centres inhibit themselves. How they can do this I must confess that I shall be unable to understand until I see Dr. Cattell get into a basket and lift that basket off the ground, or get into a wheelbarrow and wheel himself along.

Critical Digests.

ON THE MORBID ANATOMY OF FRIEDREICH'S DISEASE.

BY T. A. ORMEROD, M.D.

In the last few years, a considerable number of cases have been recorded, illustrating the clinical history of this disease, but very few observations upon its morbid anatomy. It may be well, therefore, to give a detailed abstract of three publications, in which new pathological observations are to be found.

We will begin with two cases by Rutimeyer.[1] These have been already noticed, from their clinical side in 'BRAIN';[2] having been the subject of a previous communication by Rutimeyer.[3]

CASE I.

Heinrich Kern, æt. 20.—Onset between 6 and 7 years of age, with simple ataxia first of lower, then of upper limbs. No affection of sensibility till the sixth or eighth year of the disease. Affection of speech, curvature of spine; finally, paralysis, and wasting of the legs, dorsal contraction of the great toes; diminution of cutaneous sensibility of the legs; nystagmus. Absence of patellar reflex; no affection of pupils, no pains or girdle sensations. Death from "general weakness," after the disease had lasted about fourteen years.

Nothing special to note in the post-mortem appearances; death was ascribed to fatty degeneration of the heart, and hypostatic congestion of the lungs.

Examination of the nervous centres after hardening in Müller's fluid shewed the brain and medulla to be uniformly wasted; the pia and arachnoid of the lumbar cord thickened and adherent; the nerves of the cauda equina and posterior nerve-roots thinned.

There was degeneration of Goll's columns throughout the cord; and in the cervico-dorsal region the inner parts of the cuneate fasciculi were involved, and in the lower dorsal region nearly the whole of them. The lateral columns were also degenerated, over areas corresponding to the crossed pyramidal and direct cerebellar tracts.

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[1] Virchow's 'Archiv,' Bd. 110, p. 215.
[2] 'BRAIN,' No. XXV. Table I., No. 7. Table II., Nos. 18 and 20.
[3] Virchow's 'Archiv,' Bd. 91.

Measurements of the cord after hardening—

In mid-cervical region		14 × 8	centim.
„ dorsal	„	9 × 6	„
In lower dorsal	„	9 × 6	„
In mid-lumbar	„	10 × 9	„

Microscopical Examination of the Cord.

Cervical regions.—Marked degeneration of Goll's columns, which was however, a little less intense near to (within one c.m. of) the posterior commissure.

Just exterior to Goll's columns was a tract of much less intense degeneration, broadest at the posterior periphery of the cord, and narrowing further forwards. In the upper cervical region this area of degeneration did not reach the posterior commissure; but lower in the cervical region it became broader and extended further forwards, so that only a narrow tract of white substance along the edge of the posterior horn was left unaffected.

In the lateral columns was an area of degeneration corresponding accurately to the cerebellar and crossed pyramidal tracts; the degeneration was of equal intensity through all this area; less marked than in Goll's column, but increasing in intensity from above downwards.

Grey substance practically normal; anterior nerve-roots normal; degeneration of posterior nerve-roots more marked in lower than in upper cervical region.

Dorsal region.—Almost the whole area of the posterior columns were degenerated, Goll's columns intensely; the part immediately adjacent to the posterior horns of grey matter least intensely. This widespread degeneration of the posterior columns did not begin to abate till the extreme lower end of the dorsal region was reached.

In the lateral columns, degeneration of the cerebellar and crossed pyramidal tracts was present.

In the grey matter degeneration of Clarke's columns (both cells and fibres), reaching its maximum in the lower dorsal region.

Degeneration of the posterior nerve-roots.

Lumbar region.—Diminution of the degeneration in the posterior columns; especially in their antero-lateral part (angle between commissure and posterior horns). The posterior root-zones, however, particularly in the upper lumbar region, were degenerated.

In the lower lumbar region there appeared in the columns of Goll, a small oval area, mesially situated, wherein the degeneration was less intense than at other parts of Goll's columns.

In the lateral columns degeneration of the crossed pyramidal tracts.

Grey matter mostly intact.

Still considerable degeneration of the posterior nerve-roots.

Sacral region.—Degeneration (fainter but appreciable) in the median and posterior parts of Goll's columns. Lateral columns intact.

The medulla was not examined.

CASE II.

Bertha Kern (sister of Case I.).—Note of Sept. 1882. Age 14; onset of disease at age of 7. Ataxia of limbs and of trunk; slight nystagmus; speech not affected. Little if any affection of sensation, no pain. Slight dorsiflexion of great toe. In 1883 rapid appearance of a spinal curvature. Permanent dorsiflexion of both great toes. Although extremely ataxic, the patient could get about by holding on to things. In Sept. 1884 a feverish attack accompanied by great prostration and pains in the limbs, in which she died. Typhoid was suspected, but this was not a substantiated post-mortem. Fatty degeneration of the heart was found microscopically.

The dorsal cord appeared to be small. Measurements (after hardening) of

Mid-cervical	region	12 × 8	centim.
Upper dorsal	„	10 × 6	„
Mid-dorsal	„	8 × 5	„
Lower dorsal	„	7 × 6	„
Mid-lumbar	„	9 × 6	„

The dura mater from the 7th dorsal vertebra downwards appeared infiltrated with blood, but there was no hæmorrhage into the dural sac. Slight adhesions of dura and pia, and some diffuse thickening of the pia mater.

After hardening in Müller's fluid, there could be seen (naked eye) degeneration of the posterior columns, limited in the upper cervical region to part of Goll's columns, but lower down spreading out towards the posterior cornua; and in the posterior part of the lateral columns a wedge-shaped area of degeneration reaching from the mid-cervical to the lower dorsal region.

The medulla oblongata was small. The brain was normal.

MICROSCOPIC EXAMINATION.

Upper cervical region.—In the posterior columns marked degeneration of Goll's columns reaching forward to the posterior commissure. Some affection of the fasciculi cuneati, chiefly in their middle third. Degeneration in the lateral columns, less marked, corresponding to the joint area of the cerebellar and crossed pyramidal tracts, and uniform in degree over all this area.

Grey substance normal; some loss of fibres in the posterior nerve roots.

Middle and lower cervical regions.—Goll's columns most affected. In the fasciculi cuneati the degeneration spread outwards so as to leave intact only their anterior parts, and the parts immediately abutting on the posterior cornua and posterior nerve roots.

Grey substance normal; posterior nerve-roots highly atrophied.

Dorsal region.—Still further degeneration in the fasciculi cuneati; their anterior and exterior parts, which were free in the cervical region, being now encroached upon. Low in the dorsal region, though the anterior parts of the fasciculi cuneati were involved, the stripe along the posterior grey horns, and a little triangular patch at the entry of the posterior nerve-roots were more healthy than the neighbouring parts.

In the lateral columns was degeneration of the crossed pyramidal and cerebellar tracts (as in the cervical region): but it is noted that in the upper dorsal region (and here only) an additional area was involved, reaching forwards from the anterior part of the cerebellar tract, along the periphery of the cord, to the level of the forward termination of the anterior cornua.[1]

Clarke's columns degenerated, especially at the lower part of the dorsal cord; the rest of the grey matter intact.

Posterior nerve-roots diseased.

Lumbar region.—Posterior columns; anterior parts of cuneati fasciculi normal again, while of their posterior parts a portion was affected, viz., the inner half of the area defined by Westphal as the zone of entry of the posterior roots; Goll's columns degenerated; the little oval zone of comparatively healthy substance which was mentioned in Case I., appeared in this case also. Lateral columns: less extensive degeneration, corresponding to the diminishing crossed pyramidal tract.

Degeneration of Clarke's columns in dorsi-lumbar region. Decrease in the degeneration of the posterior nerve-roots.

Sacral region.—Posterior columns: only the postero-median parts diseased. Lateral columns: degeneration of crossed pyramidal tracts still traceable.

The medulla oblongata could not be thoroughly examined; but degeneration could be traced in the funiculi graciles and cuneati to the upper part of the decussation.

A certain degree of degeneration was found in the nerve trunks (median and sciatic).

Histologically the spinal degeneration consisted (in both cases) of a growth of fine connective tissue replacing the nerve fibres; there was no special affection of vessels, and though the pia was thickened in places, there was no reason to think that the disease had spread inwards from the meninges.

The localisation of the disease moreover suggests that it was parenchymatous in origin: for it is obvious, as the author remarks, that it affected several distinct tracts of fibres, and was limited to these tracts: constituting therefore a "combined system disease." Thus in the lateral columns the crossed pyramidal tracts and direct cerebellar tracts were picked out with tolerable accuracy. (A slight extension beyond the areas of these tracts was noted in Case II.; for which we have suggested an explanation in the footnote.) This lateral degeneration was at its maximum in the lower dorsal region. The direct pyramidal tracts were not affected, Clarke's columns were diseased, which accords with the German view, that the cells of these columns are the trophic centres for the cerebellar tracts.

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[1] This probably represents the antero-lateral ascending tract.—(Abstractor's Note.)

In the posterior columns, Goll's tracts were diseased throughout; excepting a small oval centrally placed area in the lower parts of the cord, which probably forms no part of Goll's tracts. Concerning the rest of the posterior columns it may be said that in the lumbar and lower dorsal regions the disease spread widely enough to involve the posterior root-zones, while in the upper cord it was limited to the neighbourhood of Goll's tracts. How far the changes in these parts, the fasciculi cuneati, are to be considered as "systematic" degenerations is doubtful, but they correspond broadly to those of ordinary tabes.[1]

Dr. Everett Smith[2] gives a clinical account of six cases of Friedreich's disease, and the morbid anatomy of one. The clinical histories should be read in the original; we can here only make the following brief excerpts.

Five sisters were affected with the disease: three other sisters of the family did not suffer from it: five brothers were also unaffected.

The symptoms in the girls are summarised by the author as "a series of morbid symptoms, beginning at the age of 6 or 7, regular in order in progression. The first symptoms noticed were gastric disturbances and palpitation of the heart, followed by an unsteady gait which later became purely ataxic. The ataxia gradually extends to the hands; and here the disease seems to remain stationary for an indefinite length of time."

We may add the following symptoms from the clinical histories of the girls: club foot, spinal curvature, *wrist drop roughly resembling that seen in lead-palsy*, attacks of epileptic convulsions; of pain in the spine and limbs; of dyspnœa and collapse. Oddly enough, the only girl whose symptoms did not begin with cardiac palpitation, became the subject, later on, of organic disease of the heart from rheumatism. In another girl the tendon reflex[3] persisted after the gait had become ataxic, though it disappeared subsequently.

The father of these girls suffered from an allied form of paralysis, but his case differed in the following respects.

(1) The symptoms began much later in life (æt. over 60). After an attack of rheumatism, brought on by exposure to cold, there followed a "severe gastritis." But he had had attacks of nausea and vomiting for many years. He had dilatation of the heart.

(2) Although the gait was ataxic, the tendon reflexes were present.[4]

(3) The symptoms disappeared under treatment.

This man's family was decidedly "neurotic"; his father (the girls' paternal grandfather) had some doubtful ataxic symptoms; and although the father of the five ataxic girls did not drink himself, there was a marked history of drunkenness in the two generations preceding him.

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[1] For the minute localisation we must refer to the original paper.

[2] 'Boston Medical and Surgical Journal,' vol. cxiii. p. 361.

[3] Cf. a case shewn to the Medical Society.

[4] Cf. 'BRAIN,' No. 39-40. A woman with spastic paraplegia had two children suffering from typical Friedreich's disease.

The patient whose case was investigated post mortem died during one of the periods of collapse (above alluded to); it was accompanied by dyspnœa and palpitation, and vomiting so intense that she had to be fed per rectum. There had been in this case considerable paresis, in addition to the ataxy; and also affection of sensation in the lower limbs.

Post mortem, in the abdominal and thoracic organs nothing was found that bore upon the nervous disease. The muscles were generally wasted. The brain was not examined.

The spinal meninges were injected and adherent to the bony walls of the canal. The cord appeared "asymetrically atrophied, pale and in local areas softened."

The lower dorsal and lumbar regions were minutely examined, with the following results:—

The shape of the cord was altered, the posterior half being shrunken, and the left postero-lateral column looking as if it had been sliced away.

Posterior columns; their whole area intensely sclerosed, except a comparatively healthy part close to the posterior commissure. Rather more healthy nerve-fibres in lumbar than in dorsal regions.

Antero-lateral columns. Sclerosis of pyramidal tracts, crossed and direct, upon both sides, but of less intensity than that in the posterior columns.

Thickening of pia mater over the posterior and lateral columns.

Posterior nerve roots discussed; anterior healthy or nearly so.

Ganglion-cells of anterior and posterior cornua less numerous than normal, and showing degenerative changes under high powers.

Central canal blocked with round cells. In the lumbar region was a small supplementary canal just posterior to the main canal.

Dr. Pitt's case[1] is, we believe, the first reported in England in which the clinical history is supplemented by the morbid anatomy. The patient was under the care of the late Dr. Moxon on and off for seven years. He formed one of the family shewn to the Clinical Society by Dr. Gowers, and a notice of his case based on Dr. Gowers' paper appeared in a previous number of 'BRAIN.'[2] It should be added here that choreiform twitchings formed an early and a marked feature in his case, and that during the last years of his life failure in the organs of circulation supervened, as evidenced by dilatation of the heart, obstructive disease of some of the arteries of the limbs, bronchitis and dyspnœa. Death took place rather suddenly at the patient's own home.

Post-mortem.—The heart weighed 25 oz., both ventricles were dilated and hypertrophied, the left shewed fibroid patches, the aortic valves were diseased; the radial, on one side, could not be found, and on the other, contained an organised clot. The author considers that there was obliterative end-arteritis of the small vessels.

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[1] 'Guy's Hospital Reports,' vol. xliv., p. 369.

[2] No. XXV. (Table II. No. 31.)

In the brain, the only abnormal appearance was a tough and streaky grey patch "below the posterior part of the external capsule" in the left hemisphere.

Spinal cord small on section; measurements as follows:

Upper cervical region.	Normal cord.	Degenerated cord
	13 × 10	13 × 8
33 mm. lower.	15 × 9	12 × 8
39 "	16 × 9	11 × 7
51 "	12 × 8	10 × 7
67 "	10 × 8	9·5 × 7
81 "	10 × 8	8 × 7
107 "	13 × 8	8 × 8
127 "	12 × 8	8 × 7

It was of firm consistence, and there was grey degeneration of both posterior and lateral columns.

The meninges were normal.

The nerve-roots—anterior, healthy—posterior, degenerated (confirmed microscopically).

Peripheral nerves—radial, and branch of anterior crural examined and found normal.

Microscopical examination of the cord.—Weigert's method, and aniline blue black employed).

Lower lumbar region.—Posterior columns completely sclerosed, except their anterior fourth, which is healthy, and a thin strip extending therefrom backward in the middle line. Internal radicular fibres sclerosed.

Crossed pyramidal tracts shewed loss of medullary sheaths.

Upper lumbar.—Posterior columns sclerosed, except a strip along each posterior grey horn (in the anterior half of the posterior columns).

The disease of the lateral columns reaches forward along the periphery beyond the area of the cerebellar tracts.

Lower dorsal.—Similar sclerosis of posterior columns. Also of Clarke's columns. Lateral columns much as before.

Mid-dorsal.—Posterior columns as before. More extensive sclerosis of lateral columns, involving the crossed pyramidal and cerebellar tracts; and (irregularly and slightly) the periphery in front of them. Clarke's columns affected.

Upper dorsal.—Goll's columns much sclerosed. Burdach's columns become more healthy, especially their anterior third near the grey matter.

The lateral sclerosis was most marked in the dorsal region.

In both dorsal and lumbar region there was partial disease of the nerve-fibres entering the posterior grey horn.

Lower cervical.—Goll's columns completely sclerosed; slight sclerosis of posterior two-thirds of Burdach's columns.

Sclerosis of crossed pyramidal tracts less; cerebellar tracts still affected; peripheral sclerosis well marked, extending up to and slightly along the anterior fissure.

First cervical.—No degeneration in the lateral columns.

In the medulla.—Sclerosis of funiculus gracilis; slight disease of funiculus gracilis, and of funiculus rotundus.

At the level of the decussation of the pyramids two canals were visible.

The author's summary may be given thus.

1. Spinal cord extremely small.
2. Extreme sclerosis of Goll's columns, through its whole length.
3. Burdach's columns severely sclerosed, their posterior portion particularly (the disease being rather less intense high in the cord). But the strip of Burdach's columns which bounds the posterior horn and root, especially along the anterior half, is free.

In connection with (3) slight disease of funiculi cuneati and rotundi in medulla?

4. Sclerosis of crossed pyramidal tracts, most marked in dorsal region (?), not traceable above decussation of pyramids. Much less intense than sclerosis of posterior columns.
5. Sclerosis of cerebellar tracts, also not intense.
6. Slight irregular peripheral sclerosis anterior to (5); sometimes reaching along the anterior fissure.
7. Degeneration (here and there) of Clarke's columns; of posterior nerve-roots, and fibres in the posterior horns.

The localisation of the degeneration in these cases throws some light on its nature: this must be our excuse for the above lengthy abstract. All previous cases agree in this, that the posterior columns have been deeply sclerosed; and generally in this way: that the columns of Goll (postero-median) have been affected through the whole cord, the columns of Burdach (postero-external; fasciculi cuneati) extensively affected in the lower dorsal region, and less extensively higher up the cord. Here therefore the degenerative changes roughly correspond to those of ordinary tabes, and if the latter be regarded as a "systematic sclerosis," so also (quoad the posterior columns) must Friedreich's disease.

But as regards the lateral sclerosis, there has hitherto been some doubt whether this follows definite nerve-tracts, developing in them independently of the posterior sclerosis, i.e., whether it is primary and systematic; or whether it is caused by irritation spreading from the diseased posterior columns, i.e., is secondary and diffuse. Now the present cases speak strongly in favour of the former view. For in all of them the degeneration picked out known tracts of fibres, the crossed pyramidal and cerebellar in the first two, the crossed and direct pyramidal in the third, the crossed pyramidal and cerebellar in the last. In the last case also an irregular peripheral sclerosis extended round the anterior parts of the cord in its upper parts. This is reasonably ascribed to disease of the antero-lateral tract (Gowers, Hadden, Tooth); an explanation which may account for the existence, in other cases of combined sclerosis, of a peripheral degeneration not corresponding to the cerebellar tracts. And since there is no evidence that the disease spread from the posterior to the lateral columns, either by contiguity or in other ways,[1] the inference is that the process has been one of independent degeneration in several nerve-tracts; the antero-lateral sclerosis probably being secondary to the posterior in point of time, but in no other sense.

Further, the mere localisation in tracts already marked out by development and function argues powerfully for the view, that the disease is ab initio parenchymatous and begins in the nerve-fibres. Dr. Pitt advances the view, that this nervous degeneration is itself dependent upon hereditary disposition to disease in the blood-vessels; for he finds an objection to what may be called the purely nervous view in the fact, that the most sclerosed areas (Goll's columns) are just those which develop later than the less sclerosed (crossed pyramidal tracts). But we hardly think it proven that the latest in development must necessarily degenerate first; for other factors may interfere with the working of such a law: e.g., as Dr. Pitt himself indicates, the posterior columns may break down first because they have more continuous work thrown on them. And, clinically, the vascular phenomena seem to have appeared late in Dr. Pitt's case; while some of those symptoms which he singles out in other cases as vascular, e.g., headache, vertigo, comatose attacks, may just as well have been nervous. But his remarks on the condition of the circulatory system are none the less valuable; for whatever their relation to the nervous disease, symptoms from this side must be of high prognostic importance.

A word may be added on the nomenclature of the disease. Dr. Everett Smith,[2] taking as text Dr. Gowers' remarks on the inconvenience of naming diseases after individuals, proposes for it the name "postero-lateral spinal sclerosis of generic origin." The term "generic" is ingeniously chosen to express a family grouping of cases, necessarily connoting transmission as "hereditary" seems to do. As a shorter name for ordinary use Dr. Smith proposes "generic ataxia." But here he would, we presume, be met by Dr. Pitt, who says that "the pathology and course of this disease and of ordinary locomotor ataxia are entirely distinct, the only common symptom being the loss of the knee-tap." We do not altogether subscribe to this latter statement, but at least it shews that till our views about a disease are thoroughly stereotyped it is difficult to find for it a rational name.

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[1] It should be mentioned, however, that Dr. Everett Smith notes in his own case an inflammatory condition of the cells of the central canal like that observed by Brousse, and believed by him to represent the channel by which the disease spread from one part of the cord to the other. Dr. Smith's own opinion is that it spreads by means of meningitis. Seeing that the antero-lateral sclerosis in his case exactly corresponds to the crossed and direct pyramidal tracts, we hardly see how this can be maintained.

[2] 'Boston Medical and Surgical Journal,' March 1, 1888

CHRONIC HEREDITARY CHOREA.

BY W. P. HERRINGHAM, M.D.

In 1842 Dr. Waters of Franklin, N. Y., wrote to Dunglison describing a peculiar form of chorea infesting certain families in that neighbourhood, which was remarkable in four points. It was markedly hereditary, it rarely appeared before adult life or after 45, it was incurable, and it in all cases induced dementia. Dunglison inserted the letter in his third edition, with the remark that he had never himself seen anything answering to this description.

The same disease was found by Dr. Charles Gorman of Luzerne, Penn.,[1] to prevail in other portions of the country also.

The next description is by Lyon,[2] who writes twenty years later that he has been familiar with it all his life, that it occurs in certain families only, that it is hereditary, and that it is called "migrims" by the people. He does not describe the disease in any detail, does not mention any mental affection as occurring with it, and in several instances states that it began in childhood. He gives three histories: (1) in which five generations, (2) and (3) in which three generations, were affected. Case No. 1 had two brothers who never had chorea, but one of them had two children "in whom well-defined chorea has existed for many years." He says, "To these cases many more might be added."

In 1872 Huntington[3] described a chorea hereditary in a few families in a part of Long Island. It affected males more than females, came on gradually, always after middle life, was incurable, and was usually accompanied by insanity and a tendency to suicide. If one of the family escaped, his descendants were ever after free from it.

Ewald[4] in 1884 described two cases in different families. There was the same strong hereditary character, but in neither case was the mind affected, nor does he mention insanity in any of the affected relations.

Clarence King[5] gives a family history of disease in four generations and a description. He lays stress upon the insanity which accompanies it.

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[1] 'Inaug. Dissertat. at Faculty of Jefferson Med. Coll. of Philad.,' quoted by Dunglison.

[2] 'Amer. Med. T.,' N. Y., 1863, vii. 289.

[3] 'Med. and Surg. Reporter,' Philad., April 13, 1872, quoted by Sinkler in Pepper's System, vol. v. 440.

[4] 'Zeitschrift f. klin. Med.' 1884, vii., Suppl. H. 51.

[5] 'N. Y. Med. Journ.,' xli. 468, 1885.

Peretti[1] gives an elaborate family tree of his case; the whole family springs from Frau N., who was choreic. Two children were healthy, and had healthy descendants; two were diseased. They had between them fifteen children, of whom nine had the disease and four had other nervous affections. Of the nine choreics, one had four children over 30, none of whom were affected, and three of these had healthy children themselves; four others had children, among whom one male aged 23 and probably two boys were already diseased. Insanity is only mentioned in four of the nine.

Huber[2] had a case of a man aged 38: the disease began at 30 in his face, spread first to the arms, then to the legs. He notices that the movements, which otherwise resemble juvenile chorea, are easily controlled. The only mental symptom in this case was weakness of memory. The man's sister, aged 42, was choreic and demented; his father choreic and insane, his father's sister choreic. The disease was found occurring in four generations.

In the Collective Investigation Report[3] compiled by Dr. S. Mackenzie, West mentions a family in which three generations were diseased.

Zacher[4] describes a case and gives the family history, showing three and perhaps four generations affected.

Hoffmann[5] describes another set of relations similarly diseased.

Macleod[6] treated and dissected two sisters, who were choreic and insane; their father had died of a disease which made him shake all over, and two brothers are said to be choreic. It is to be hoped that more details will be forthcoming; for though the cases seem probably to belong to this category, they are exceptional in more than one point, and the family history is insufficient.

Lannois[7] traces the disease in four generations of one family, and describes six of the cases at length.

Ewald, Huber, Zacher, Hoffmann, and Lannois give most careful and excellent descriptions of the cases they observed.

From these authors a good picture of the disease can be constructed. It is a disorder of movement, affecting the voluntary muscles only, which exactly resembles chorea in appearance. Hoffmann does indeed describe a case (Jos. Kärcher) in which the movements were more like violent tetanic spasms; but it is so different in other respects also, that with all deference to Hoffmann I do not consider it right to class it with the present group. The disorder is hereditary, and affects more individuals of the families which it infests than most other hereditary diseases. Thus, taking only the patient's own generation, King has four affected out of five, Peretti nine out of sixteen, Huber two out of two, Hoffmann five out of nine (Wipfler family). In the second generation of Lannois' family five out of twelve were choreic. These five had fourteen children, of whom eleven were diseased. On the other hand, all agree that when once the chain is broken the disease does not recur. An exception to this is, however, to be found in Lyon's paper; but he does not seem to be speaking from personal knowledge. The character of the inheritance is therefore peculiar. The disease affects both sexes, and is heritable either through the female as in Ewald's, or through the male as in Huber's case. It nearly always begins at or after 25 years of age, but occasionally earlier. Lyon, an exception in this as in many other points, speaks of its beginning at puberty. Peretti has a man of 23 in whom it had already begun to show itself, and another who had twitchings from childhood, and died paralysed at 19; while Felic. Wipfler (Hoffmann) began to be diseased at the end of her school-days. Twitchings in the face are noticed in children of diseased families by Peretti and Zacher. Waters places the age of onset not before adult life or after 45; Huntington says it never comes on until middle life; Ewald's cases nearly all began at 30; King places the onset between 25 and 35; Macleod's cases began at 60 and 70.

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[1] 'Berl. klin Woch.,' 1885, No. 52.
[2] 'Virch. Arch.,' cviii. 1887, 267.
[3] 'Brit. Med. Journ.,' 1887, i 425.
[4] 'Neurolog. Centralblatt,' 1888, 34.
[5] 'Virch. Arch.,' 1888, 513.
[6] 'Journ. Mental Science,' xxvii. 1882, 191.
[7] 'Rev. de Méd.,' vii. 1888, 645.

The disease does not develop out of ordinary chorea; but begins without known cause by twitching in the face (King, Huber), spreading thence to the arms and later to the legs, or is general (Hoffmann) from the first. The movement is violent, and coarse in character, not like Friedreich's paramyoclonus, a fibrillar twitching insufficient to move the limb. In the legs it produces a curious gait: there is a sudden stoppage, the patient looks as if he was going to fall forward, sways his body violently, at last is able to take a few rapid steps, and so recovers his balance (King, Hoffmann, Lannois). It is stated in several cases that coarse motor power is preserved. In Felic. Wipfler it was but small. It is not uncommon for ancestors to be described as having been "paralysed." In some cases the finer movements are impossible, but it is noticed by several that these patients are much more able than ordinary choreics to control the involuntary movements by the will, so that those who are in constant and violent movement can yet drink, eat, and dress themselves without difficulty. In most cases the movements cease in sleep, but Hoffmann noticed them even then.

There is no wasting of muscles, no anæsthesia; the deep reflexes are natural or somewhat increased, and the electrical reactions are normal.

There is no heart disease connected with the affection, nor is rheumatism an antecedent. The bodily functions are carried on naturally, and micturition and defæcation are, so long as the mind is sound, properly performed.

The disease is very commonly followed after a short time by some mental degeneration. This is noticed by every author except Lyon and Ewald. Though usual, it is not invariable. Two of Huber's cases were intelligent officials up to their death, and Ewald's cases were both perfectly sane. It not unfrequently happens that other neuroses occur in members of the family who are not choreic (Peretti, Huber, Lannois). The choreic insanity begins with loss of memory and childishness, and goes on gradually to dementia. In two cases (Huber, Zacher) there were fits of violent destructiveness.

There have been three post-mortems recorded. Macleod (if his cases be admitted) found in one case a subdural hæmorrhagic cyst, with leptomeningitis over one side of the brain only, in another multiple tumours in the dura mater. Huber transcribes the post-mortem record of the father of his own patient. Pachy- and leptomeningitis were the only lesions. Another post-mortem, which will be of great importance, is to be expected from Klebs, who has dissected one of the cases which Huber reports in detail.

It is of the greatest interest to compare this disease with ordinary chorea. The movements are of similar character, and in both there is commonly mental affection. The one is closely connected with rheumatism and heart disease, and recovers. The other has no such connection and is progressive. The conclusion which the comparison suggests is that the two are produced by affections of the same parts, but of a different nature. On the other hand, it has certain analogies with general paralysis. Tremor and weakness in the one are replaced by loss of control and involuntary movement in the other; loss of muscular power supervenes at the end of some cases. The exalted delusions of the general paralytic are in some instances (Hoffmann) of this disease represented by cheerful misrepresentation of their real condition. The post-mortems show here, as in general paralysis, affections of the meninges.[1]

That it is an affection of the brain is a generally expressed opinion, and that it is impossible as yet to say more is obvious.

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[1] De Witt, 'Cincin. Lanc and Observ.,' 1876, xix. 408, has a paper on bilateral chorea occurring in a general paralytic. I have not seen it, but it sounds as if it formed a link.

W. P. HERRINGHAM.

Abstracts of British and Foreign Journals.

Current Nerve Anatomy and Physiology.—Titles and Indication of Contents of recent Papers. By ALEX. HILL, M.D., Master of Downing College, Cambridge.

TEXT-BOOK.

1. Petit Atlas Photographique du Système Nerveux. J. LUYS (*one vol.*, 16*mo*, *Paris*, *T. B. Baillière*, 1888).

COMPARATIVE ANATOMY.

2. The Nerves and Vessels of Ammocoetes and Petromyzon Planera. A. DOHRN (*Mitth. aus der zool. Station zu Neapel*, viii. 2. pp. 233–307, *July*, 1888).
3. The Comparative Anatomy of the Brain. The Fore-Brain. EDINGER (*Abhandl. der Senckenbergischen Naturf. Gesellschaft*, 1888. *Separatabdruck*, *Frankfurt Diesterweg*; 4 coloured lithograph plates).

The disposition of the mantle, and the arrangement of those bundles of medullated fibres which are common to all vertebrate brains investigated by means of section series. Staining with Weigert's hæmatoxylin and carmine, or acid fuchsin.

In all transverse sections, two portions are to be considered; the thick ventral portion projecting up into the floor of the ventricle, and the thin roof and outer wall. The former is the corpus striatum or stem-ganglion, the latter the mantle. The mantle is always covered with a layer of grey matter, but a cortex in the ordinary sense is a late development in the animal series. Part of the mantle in cyclostomes and teleostei, is devoid of cortex, like the septum pellucidum of primates. The stem-ganglion is always nervous.

1. Ammocoetes—little round cells in small groups uniformly disposed through the whole thickness, separated by finely-fibrous substance which arranges itself into thicker fibre bundles, forming in front, a commissure and an olfactory chiasm, behind a commis-

sure (C. interlobularis), and a track running towards the mid-brain, but not ending there. The author regards it as a veritable basal fore-brain bundle. The fine plexus fibres take Weigert's hæmatoxylin almost as well as ordinary medullated nerves, although not surrounded by what we ordinarily regard as medullary sheaths.

2. Teleostei.—The mantle is so thin as to be usually overlooked. It presents no median fold similar to the longitudinal fissure of higher vertebrates. In front of the mid-brain it forms the sack-like recess for the pineal gland, owing to the great development of the mid-brain; the 'tween-brain is obscured, and has often been overlooked, but it has the usual ganglia and bundles of fibres. From the stem-ganglion a single medullated basal fore-brain bundle (pedunculus cerebri) originates. In developing Corvina it can be seen to originate by three roots, two from the more thickly grouped cells in the dorsal and lateral region, the third from a well-defined group near the median line.

In all vertebrates there is a commissure (or chiasm) between the basal fore-brain bundles in the 'tween-brain region. The commissura interlobularis (Ant. Com.) was usually non-medullated; some of its fibres unite the stem-ganglia, others come from the olfactory nerves. The crossed fibres are prolonged into the 'tween-brain. The stem-ganglion represents only the putamen and caudatus of higher animals, not the globus pallidus. It gives origin to fibres and is not intercalated in their course. In the same way the basal fore-brain bundle represents only that part of the pedunculus cerebri which springs from the corpus striatum.

3. Selachians.—The fore-brain varies very much in form, but usually presents a central fused mass with an unpaired ventricle, and two enormous lateral olfactory lobes. The homologies of the central mass are very obscure. To this problem the author devotes himself. By studying embryos it is found that the fore-brain consists almost entirely of anterior wall, this is greatly thickened and folded down anteriorly, constituting the primary unpaired fore-brain which alone exists in the rays; the ventricle disappears. From the primitive cerebral cavity, frontal-lobes bulge out in the sharks.

In adult cartilaginous fishes the fore-brain exhibits three groups of cells beside the nerve cells everywhere scattered through its substance. The dorsal stem-ganglion group gives origin to the basal fore-brain bundle; the ventral group to the anterior commissure, and perhaps to the olfactory nerve. In the "most occipital portion of the mantle" lies a third group which gives origin to a "mantle bundle." The basal fore-brain bundle passes backwards, some fibres cross to opposite sides; some fibres end in the thalamus, others go on to the medulla oblongata. The mantle-bundle crosses to opposite side at base of fore-brain. This crossing has nothing to do with the often described commissura Halleri. The anterior commissure which connects the stem-ganglia contains but few medullated fasciculi in the rays; in Scyllium it is reinforced by considerable medullated tracts from the olfactory lobes. There is in addition a median bundle originating in front and dorsally, and passing downwards to the basal fore-brain bundle.

The amphibian brain is the simplest in the vertebrate series. By the backward prolongation of each cerebral hemisphere, occipital lobes are formed. The nerve fibres do not become myelinated until just before adult life; the basal longitudinal bundle passes backwards towards the medulla, some fibres, however, end dorsally in the 'tween-brain, and others in the regio infundibuli.

In reptiles an indubitable brain-cortex is met for the first time (there are probably in some part of the mantle both of amphibia and dipnoans, cortex formations). Author gives a description of this as found in different parts of the mantle and in different animals; it appears that the typical cortex formation only occurs in the occipital region, and at the dorsal angle of the median surface. Scattered round pyramidal cells are found on the outer wall of the cortex, in relation with fibres of origin of the olfactory nerve. A third kind of formation is found between the other two.

CORTEX.

4. Comparison of the Convolutions of the Seals and Walrus with those of Carnivora, and of Apes and Man. TURNER (*Journal Anat. and Phys.* xxii. pt. 4, pp. 554–582, *July* 1888).

Contains a repetition of work done for the Challenger Reports, including a detailed description of the brains of a large number of carnivora other than the Pinnipedia. Treats of the homologies of these brains among themselves, and with those of primates, especially the limits of the hippocampal lobe, and the Island of Reil which the author regards as situate on the general surface in carnivora, as the whole, or part of the Sylvian convolution. In the polar bear an entire arched convolution is concealed within the fissure of Sylvius; the Sylvian convolution is partially concealed in the seals and walrus. This explains the apparently discrepant results which follow stimulation of the margin of the fissure of Sylvius in monkeys and carnivora. Author reviews the evidence as to the homology of the fissure of Rolando and crucial sulcus, and inclines to the belief that the former is represented by the coronal fissure of carnivora, and that the latter is of secondary value, and not necessarily represented in higher animals. The præsylvian fissure is the homologue of the præcentral; the intraparietal fissure is to be found in the anterior part of the lateral (second curved) of carnivora.

LOCALISATION.

5. Communications on Localisation in the Great Brain, and their Practical Value. JASTROWITZ (*Deutsche Med. Wochenschr. No.* 5, 1888).

Calls attention, more especially from the surgeon's point of view, to the number of difficulties and uncertainties which still stand in the way of diagnosis of brain lesions. Calls attention especially to two cases, in the first of which hemiamblyopia was associated with one lesion at the upper end of the ascending frontal convolution, and another at the junction of the gyrus fornicatus and præcuneus. In the second case no motor disturbance resulted from a tumor as large as a cherry in the ascending parietal convolution.

CORTEX—STIMULATION.

6. Landois' Experiments with Chemical Stimulation of the Brain. LENBUSCHER and ZIEHEN (*Centralbl. f. Klin. Med. No.* 1, 1888).

CORTEX—EXCITABILITY.

7. Co-existence of Inexcitability of the Excito-Motor Zone with Persistence of the Motor Functions of this Nervous Centre, and also with the Production of Violent Epileptiform Attacks. BROWN-SÉQUARD (*Comptes Rendus Soc. de Biol.* p. 354, *April* 14, 1888).

After considerable loss of blood the cortex becomes, as is well known, inexcitable. An animal in this condition, however, will move about normally without showing motor or sensory disturbance. Stimulation of its cortex will sometimes throw it into convulsions.

TEMPERATURE OF CEREBRUM.

8. Temperature Changes in the Great Brain associated with Emotions. TANZI (*Phys. Centralbl. No.* 3, pp. 57–64, *May* 12, 1888).

Thermo-electric investigations carried out in Schiff's laboratory at Florence.

Six dogs and two apes experimented upon; anæsthesia with morphia in the circulation and cocain applied locally. The thermo-electric needles sunk through trepan-holes until they touched or perforated the dura, but did not injure the brain. Various suggestive sensory stimuli were applied, and the place, time, and character of the alteration of temperature recorded. When the narcosis was too deep for changes in the brain, variations in the temperature of the spinal cord were observed, which sank to very small proportions when the brain variations began to set in. The thermal changes which resulted from "mind stimuli," whether on one or both sides of the body, affected the whole brain on both sides. They were not altered by faradisation of the sciatic nerve, or placing ammonia under both nostrils. The changes did not consist in a single elevation or depression of temperature, but in an irregularly rhythmic change. In the spinal cord the rhythm kept pace with the reflex movements; in extreme cases the variation reached 3° C. above or below the normal. The changes, judging from the effects of different stimuli, are proportional to the intensity of the emotion. They appear to be independent of change in circulation.

CORPUS CALLOSUM.

9. Cases of abnormally short Corpus Callosum Cerebri. SCHROETER (*Allg. Zeitschr. f. Psychiatrie*, xliv. *Nos.* 4 and 5, p. 408, 1888).

EPIPHYSIS.

10. Epiphysis Cerebri in Petromyzon fluviatilis. WHITWELL (*Journal of Anat. and Phys.* xxii. pt. 4, p. 502, pl. xviii., *July* 1888).

Corrects in certain points the description published by Ahlborn.

CEREBELLUM.

11. Constituents of the Anterior Peduncles of the Cerebellum. BECHTEREW (*Archiv f. Anat. u. Phys.*, 1888, *Anat. Abth.* 2–4, pp. 195–205, pl. xiii., *June* 15).

Commences with historical account. Developmental method. Human embryos. Not fewer than four tracts can be distinguished by the periods at which their fibres become myelinated; *1st.* (in fœtus 27–28 cm: long) has a shorter course than the others; *2nd.* (at 33 cm.) is longer and lies in the dorsal portion of the half-moon shaped peduncle as seen in section carried between the cerebellum and corpora quadrigemina; *3rd.* (35–38 cm.) smaller than the two former, lies in the middle of the half-moon; *4th.* and longest (at end of intrauterine life) lies on the inner side of the others. The first is isolated from the others both at its origin in the cerebellum, and at the decussation; its fibres do not go into the cerebellum itself, but are lost in the neighbourhood of the nucleus nervi vestibularis (Bechterew) on the outer side of Deiter's nucleus. Forwards they only travel with the others as far as the front of the pons where they cross the middle line. The remaining three tracts connect the cerebellum with the great brain; the crossing seems to be complete. All three go into the red nucleus, but whether their fibres go round its cells, or are broken by them, undetermined. The second and third tracts are connected with the central nuclei and the cortex of the vermis, that is to say with the region connected also with the fibres of the posterior peduncle, except those coming from the under olive. Comparing their development with that of the tracts which make up the posterior peduncle, it is seen that the dorsal bundle (2) myelinates at the same time as the direct cerebellar tract; the middle at the same time as the fibres on the inner side of the corpus restiforme which originate in the nucleus nervi vestibularis, and the fibres from the nucleus of Goll's column.

CIRCULATION.

12. The Brain Arteries and their Enclosure in Bony Canals. RUDINGER (*Archiv für Anat. u. Physiologie*, 1888, *Anat. Abth.* 2–4, *July* 15, pp. 97–109, pt. vi.).

The diameter of the carotid arteries is not much more than one half that of the carotid canal. The relation of the vertebral arteries to the foramina in the transverse processes is about the same. The rest of the space is occupied by the venous network. The pulsation in the arteries is a factor in propelling the blood in the veins; the arrangement of the veins cannot regulate the blood supply to the brain.

CIRCULATION.

13. Lymph-paths of the Brain. ROSSBACH and SEHRWALD (*Centralbl. f. d. Med. Wissensch.* 26 and 27 *June*, 1888).

The conclusions in this paper are based entirely upon the supposition, in which the authors considered themselves justified, that the method which Golgi trusts to for staining nerve-cells and their processes is really a method for filling up the periganglionic and perigliac spaces with silver precipitate. Interpreting the appearances which result when Golgi's method is used in this light, the authors give an account of the arrangement of the smaller lymph spaces and tracts in the brain. The figures, they say, which Golgi interprets as stained ganglion cells are always larger than the cells from the same piece of tissue as shown up by other staining methods. The form of the cells is different. No encircling space, such as is always to be seen with other methods, is visible around the dark figures. When Golgi's incrustation-mass is dissolved in ammonia the large figures give way to small cells of usual appearance. When the staining is incomplete pieces of axis cylinder, etc., are seen in periganglionic spaces. The well-known uncertainty of Golgi's method depends upon the varying condition as to size and fulness with lymph of the lymph-tracts. The lymph spaces in the retina, stomach and elsewhere can be filled with precipitate of bichromate of silver by Golgi's method.

CIRCULATION.

14. The Influence of Simple and Epileptogenous Excitations of the Brain upon the Circulatory Apparatus. FRANÇOIS-FRANCK (*Comptes Rendus Acad. des Sciences, Paris,* cvii. 5. *July* 30, 1888).

CRANIAL NERVES—TASTE.

15. The Course of the Taste Nerve Fibres to the Brain, SALOMONSOHN (*Inaug. Diss., Berlin,* 1888).

Case of loss of taste over anterior two-thirds of tongue on one side, associated with loss of smell, blindness, and protrusion of eye-ball on same side. Diagnosis of a syphilitic tumor extending from foramen opticum to foramen rotundum. Conclusion that taste fibres of lingualis passed via chorda, geniculate ganglion, great superficial petrosal, spheno-palatine ganglion to superior maxillary nerve.

SPINAL CORD—DEGENERATION.

16. Lesions of the Spinal Cord produced by passing Anæmia of this Organ. SPRONCK (*Archives de Phys.* xx. pt. 1, p. 1, 1888).

Temporary ligature of the abdominal aorta induces permanent degenerations which only affect primarily the ganglion cells and their processes. The degenerations of the medullated fibres are secondary.

SPINAL CORD—NUTRITION.

17. Changes in the Spinal Cord which follow Occlusion of the Abdominal Aorta. SINGER (*Sitz. der k. Acad. d. Wiss.* xcvi. pt. 3, p. 136, 1888).

The aorta was tied below the renal arteries in rabbits. Permanent sensory and motor paralysis usually resulted. The spinal cord was examined after varying intervals with the Weigert-Pal method. Almost all the cord degenerated with the exception of the posterior columns, roots and ganglia. The degeneration of the cells of the anterior brain was obvious.

SPINAL CORD—FILUM TERMINALE.

18. The Anatomy of the Spinal Meningies, the Sacral Nerves and the Filum Terminale in the Sacral Canal. TROLARD (*Archives de Phys.* ii. 6, pp. 190–200, *August* 15, 1888).

The arachnoid cavity is not continued to the extremity of the sacral canal, but terminates at the second sacral vertebra. The first four posterior branches of the sacral nerves form a plexus giving rise to a posterior nerve. The last two in like manner constitute cutaneous and musculo-cutaneous branches, etc.

REFLEX ACTION—KNEE-JERK.

19. The Reinforcement and Inhibition of the Knee-jerk. BOWDITCH (*Boston Medical and Surgical Journal, May* 3, 1888).

Reflex Action—Respiration.

20. A Reflex Action on Respiration of Stimulation of the corneal branches of the Trigeminus. Guttmann (*Virchow's Archiv*, xciii. pt. 2, pp. 222–229, *August* 1888).

Boy of four years old poisoned with atropin—unconscious. Irritation of cornea with finger caused cessation of respiration in expiration lasting 5·9 seconds.

Senses.

21. A Kinesiæsthesiometer, with Remarks on the Muscular Sense. Hitzig (*Neurolog. Centralbl. Nos.* 9 and 10, 1888).

Method depends upon the measurement of the acuteness of the muscular sense by means of pieces of wood of the same size, but differently weighted with lead. The author finds that the ability to appreciate differences in weight sinks with the depression of general sensation in the limb, a conclusion which is opposed to the central apperception of the force discharged to do muscular work.

Peripheral Nerves.

22. Electric Organ of the Skate. Sanderson and Gotch (*Journal of Physiology*, vol. ix., Nos. 2 and 3, pp. 137–167, *August* 1888).

Histological account with figures. Authors do not believe in the nervous nature of the branched cells which are in relation with the terminal ramifications of the nerves. Minute and careful analysis of the electric phenomena of the shock, one hundredth of which is sufficient to throw the image off the scale of the electrometer, the maximum electromotive force being about half a volt. The current passes along the tail from before backwards.

Electric Organ.

23. Remarks on some New Researches on the Torpedo. E. du Bois-Reymond (*Sitz. der K. P. Akad. zu Berlin*, xxii. p. 531, 1888).

A criticism of the paper in Proc. Roy. Society (vol. xlii. p. 357, 1888) by Gotch.

PERIPHERAL NERVES.

24. On the Distribution of the Cutaneous Nerves on the Dorsum of the Human Hand. BROOKS (*Internat. Monatschr.* vol. v. pt. 8, 1888).

By means of dissections of the skin, with nerves attached, under water, the author shows that the nerves of the hand cross one another much more extensively than is usually supposed. Skin areas are supplied by more than one nerve. Hence partial retention of sensibility after section of particular nerves.

PSYCHOLOGY.

25. The Memory of Muscular Sensations. BEAUNIS (*Rev. Philosoph. No.* 5, p. 569, 1888).

HISTOGENY—OPTIC NERVE.

26. The Histogeny of the Retina and of the Optic Nerve. FALCHI (*Archiv fur Ophthalmologie*, xxxiv. 2, pp. 67–108, 3 plates, *July* 20, 1888).

A detailed account of the manner and date of formation of the several layers of the retina, etc.

HISTOGENY.

27. Development of the Substantia Gelatinosa Rolandi. CORNING (*Archiv f. Mikrosk. Anat.* xxxi. pt. 4, *June* 14, 1888, 4 woodcuts).

The object of the investigation is to determine; (1) from which part of the original neural canal the substantia gelatinosa develops, or whether it is formed, as His believes, not from the neuro-epithelium at all, but from cells which have wandered into the neural tube from outside; (2) how soon it is distinguishable from surrounding parts; (3) whether the development throws any light upon the contrast which its structure presents to that of other parts. The author finds that it first appears as a local thickening of the inner layer of the grey matter in its dorsal portion. This thickening extends outwards on the dorsal surface towards the point of entrance of the posterior roots. It finally divides into one portion, which remains in the dorsal wall of the central canal, and the portions which permanently occupy the apices of the posterior horns. In new-born animals the substantia gelatinosa consists of two kinds of cells. The first kind have large clear-staining nuclei with oval or spindle-shaped cell-bodies. The second kind have darkly staining nuclei and indistinguishable cell-bodies. This distinction into one class resembling nerve-cells, and another class resembling glia-cells, is still more apparent fifteen days after birth. The substantia gelatinosa comes from the cells which form the dorsal portion of the "inner plate." They do not lose their indifferent character until after both the anterior-horn cells and the grey matter of the posterior horn are differentiated.

TERATOLOGY.

28. The Brain and Eyeball of a Human Cyclopian Monster. CUNNINGHAM and BENNETT (*Trans. Royal Irish Acad.* xxix., 1888, pl. 4 and 5).

Full term female child. No other deformities. Single lozenge-shaped opening, four eyelids. Mid and hind brain natural except no brachia to corpora quadrigemina, and no mesial geniculate bodies. 'Tween-brain distended, but epiphysis normal (although hollow); optic thalami fused; pulvinar absent. Single optic nerve arising from the base of the brain in front of the tuber cinereum. Fused cerebral hemispheres, the margin of the cortex above the mid-brain being raised into a narrow ridge ending on either side in a wider elevation corresponding to the fascia dentata. Corpus callosum, anterior commissure and fornix absent. Fused cerebral hemispheres terminated in front in a knob of cerebral matter about the size of a small bean protruding through frontal bone to bottom of left nasal pit. Authors look upon this as indubitably fused olfactory lobe. Optic nerve was single to its termination, but there were two retinal expansions forming two chambers within the eyeball. All the parts in front of the retina were double.

METHOD.

29. Carmine Staining for Nerve-tissues. UPSON (*Neurolog. Centralbl.* ii. p. 319, *June* 1, 1888).

Three methods; (1) 5 c.c. Grenacher's carmine alum (1 gramme carmine boiled in 100 c.c. 5 per cent. solution of alum), 10–20 drops of acetic acid, 1–3 drops phospho-molybdic acid, stains nerve-cells, axis cylinders and connective tissue in 2–10 or more minutes: useful for over-hardened tissues; (2) carmine alum saturated with zinc-sulphate, same results; (3) water 4, alcohol 1, "carmine acid" 0·06, sections immersed 3–10 minutes. Colour fixed with acetic acid (orange), saturated lead acetate (blue), sulphate of iron (black), manganese sulphate (red), nickel sulphate or barium chloride (violet). Axis cylinders, nerve cells, and connective tissue stained. Tissue must be well hardened.

Remarks on the foregoing, Krauss (*ibid.*).

METHOD.

30. Summary of the Osmic Acid and Gold Chloride Method. KOLOSSOW (*Zeitschrift f. Mickroskopie*, vol. v. pt. 1, pp. 50–53, 1888).

METHOD.

31. The Differences in Chemical Reactions of Nerve-cells. FLESCH (*Mittheilungen der Naturf. Gesellsch. in Bern*, 1888).

An attempt to bring together the well-known differences in the behaviour of nerve-cells to colouring reagents, metallic oxides (osmic and chromic acid), alkalies, acids, etc., with a view to classifying nerve-cells on a chemical basis. Cells grouped as chromophobic and chromophilous. Author establishes a relation between function and chemical behaviour; staining power resides in protoplasm, not in the granules. The chromophilous cells are as a rule smaller, contain dark granules, have a sufficiently alkaline reaction to yield Ehrlich's methyl blue reaction. When methyl blue is injected into the circulation of the living animal they stain like nerve fibres. They strongly reduce osmic acid. The protoplasm of the cells stains in carmine, etc., but not the nucleus (nor does the nucleus stain in chromophobic cells). Eosin stains the nuclei of all nerve-cells when they have been hardened in acetate of lead or other alkaline or neutral media. This distinguishes nerve-tissues from other tissues.

Pugibet on Paralyses in Dysentery and Chronic Diarrhœa of Hot Countries. (*Revue de Médecine*, February–April, 1888).—Out of eighty cases of dysentery and diarrhœa contracted in Tonquin, Dr. Pugibet relates *seven* in which nervous complications ensued. It is to be noted that all these patients came ashore in a state of great debility, insomuch that at least four of them died within a few days or weeks. The form taken by the "paralyses" was various. In two cases the muscles moving the head upon the trunk (excepting the sterno-mastoids) were affected, with both deltoids and, to a less extent, the flexors and extensors of the forearms and wrists. In a third there was hemiplegia, and in a fourth, paralysis of the serratus magnus on one side, and the trapezius and the rhomboidei on the other. In the three other cases there were respectively, transient *glycosuria*, double trigeminal *anæsthesia*, with partial deafness; and finally paralysis of the bladder and rectum supervening, with a small patch of cutaneous anæsthesia, only twenty-four hours (!) before death. The special features of the motor paralyses were quick onset without cerebral symptoms, short duration and recovery beginning at the parts last (which were also least) affected. Several hypotheses as to the nature and seat of the lesions are discussed. The author cannot accept *ascending neuritis*. This theory, as explaining the paralysis following after diphtheria and other acute diseases, is not now widely accepted, Vulpian and others having declared the nerve lesions actually found to be secondary to a primitive lesion in the medulla. Dismissing peripheral conditions, whether of nerve or muscle, as inadequate to cause the symptoms under discussion, it is shown that they do not follow nervous distribution. Nor can they own a reflex origin, like some rare paralyses due to uterine displacements or affections of the genito-urinary tract, for else they would observe some correspondence, in time or in severity, with the intestinal lesions. But this is not the case. M. Pugibet therefore attributes them to transient lesions of the anterior cornua of the cord; and cites two cases from Delioux in which, on autopsy, softening was discovered in the cervical and lumbar enlargements. The proneness of the post-dysenteric paralyses to affect isolated muscles, or muscular groups, or both upper or both lower limbs (according to various writers), is pointed to as a feature bringing these cases into the category of spinal affections; although it is admitted that they differ from ordinary cornual paralysis in non-febrile onset, short duration and absence of atrophy. The facial anæsthesia, deafness and glycosuria are referred to similar changes at the level of the fourth ventricle.

From the cachectic state of the patients, one might suppose that a simple failure of circulation in the nervous centres would account for the symptoms; but, on the other hand, in anæmia and chlorosis, although paralyses occur, they have not the special features seen in these cases,—sudden onset, often by night; rapid development, often a short duration and sometimes recovery coincident with aggravation of the cachexia. These diagnostic points distinguish the paralyses following a dysentery from those commonly seen in convalescents, the "asthenic paralyses" of Gubler. In the Author's view the probable explanation of them is a thrombotic coagulation in the fine capillaries of the medulla, an event favoured by the cachectic condition of the blood and by the slowing of the circulation during sleep. The effects would vary with the degree of collateral circulation established, and according to the particular group of vessels primarily affected. Thus in severe cases local atrophy or softening may follow, leading to incurable paralysis. The previous history of marsh fevers in many of the cases naturally suggests a *miasmatic* cause. In these fevers nervous complications occur, either with the accession of the fever, or in the later cachectic period. Maillot, Jaccoud and others have already regarded paralyses in the first stage as due to an early congestion of the nervous centres, whilst Rindfleisch attributes them generally to melanæmia, causing obstruction by pigment, and rupture of capillaries among the nervous elements. Whatever be the pathology, the fact remains that paralyses of paludal origin are not rare, tend to appear and disappear with the attacks of fever, commonly take the form of right hemiplegia or aphasia, and that, like other symptoms due to the same cause, they readily yield to the influence of quinine. The post-dysenteric cases do not satisfy these conditions: but it seems probable that pigmental cells (present in all miasm-infected blood) may have contributed to the capillary lesions referred to.

In addition to his own seven cases already referred to, the Author cites ten more from other physicians: and shows how in these also the seat of motor affection is very variable; that sensory symptoms are not uncommon, especially after dysentery in the acute form; that electrical contractility is normal or slightly diminished, and that as a whole the symptoms presented are ordinarily incomplete and tend to disappear in the course of a few months. In a minority of cases they may persist, or, becoming generalised, may cause death by invading the respiratory centre. For treatment,—in the paludal cases Dr. Pugibet gives hypodermic injections of the hydrochlorate of quinine, of which he has administered as much as two or three grammes within a few hours.

FORTESCUE FOX, M.D.

BRAIN.

JANUARY, 1889.

Original Articles.

HEAT-CENTRES IN MAN.

BY ISAAC OTT, M.D., EASTON, PENNSYLVANIA, U.S.

After my discovery of the seat of the cerebral inhibitory centres of sphincters, it occurred to me that by similar means I could find the location of the basal heat-centres. Transverse sections were made of the brain from before backwards, and when I divided the corpora striata, a temperature of 111½° F. was obtained.

In April 1884[1] I stated that heat-centres existed about the corpus striatum, which is true to-day. In July 1885[2] I also announced that the anterior part of the thalamus, when injured, caused an elevation of temperature due to augmented production of heat as shown by the calorimeter.

After the above dates, October 1885, Messrs. Aronsohn and Sachs[3] stated that injuries on the medial side of the nodus cursorius and beneath the corpus striatum caused an elevation of temperature with increased absorption of oxygen, increased exhalation of carbonic acid and augmented excretion of urea. It is true that they published a paper[4] (December 18, 1884, six months after my publication), on the elevation of temperature after punctures, locating the origin in the anterior part of the brain, a fact already discovered by Richet. These facts destroy the claims of Drs. Aronsohn, Sachs, and Richet to priority of discovery of the basal heat-centres.

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[1] 'Journ. of Nervous Disease,' 1884.

[2] 'Philadelphia Medical News,' 1885.

[3] 'Pflüger's Archiv,' 1885.

[4] 'Deutsche Medicin. Wochenschr.' 1884.

Further, the determination by them of the increase of oxygen absorbed and of carbonic acid thrown off, with augmented excretion of urea, was no absolute proof that the elevation of temperature was due to increased production of heat: for D'Arsonval has shown that chemical processes do not give the exact measure of the production of heat, since the incubating egg in its first stage absorbs considerable heat and also oxygen, whilst it exhales carbonic acid. This absolute proof was only furnished by the calorimetric experiments.

There have been localised in the lower animals six centres, injury to which causes increased temperature. The cruciate about the Rolandic fissure, the sylvian at the junction of the supra- and post-sylvian fissures, the caudate nucleus, the tissues about the corpus striatum, a point between the corpus striatum and optic thalamus near the median line, and the anterior inner end of the optic thalamus itself. These centres have their own laws, which serve to distinguish one from the other. In a paper published by Dr. W. Hale White,[1] there has been collected a number of cases, which I have mainly drawn upon to support the theory of localised heat-centres in man. He rules out all cases where the cause is not purely nervous. Thus cases with bed-sores, where suppuration might cause fever, are excluded.

In support of the localisation of a heat-centre about the Rolandic fissure, the following case is quite definite:—H. W. shot himself with a pistol. Upon admission he was insensible, with a small triangular wound on the right side, between the external angular process and the top of the pinna, respiration irregular, 28–35, pulse 52, strong, regular, right pupil widely dilated, left strongly contracted, both insensible to light. Almost directly after admission the bullet was extracted. Immediately after the operation, the temperature was 99·2°, but it ran up in a few hours to 104·2°. It remained high, being half an hour before death 104·4°. Death ensued twelve hours after the operation. Upon post-mortem it was found that the bullet had not perforated the dura mater. The anterior extremity of the middle lobe of the right hemisphere was severely contused, as also the third frontal convolution, the damage affecting the whole thickness of grey matter. This case lends considerable support, taken in connection with the location of the cruciate centre in lower animals, that the parts about the fissure of Rolando, when injured, cause an elevation of temperature. In confirmation of this view, a remarkable case has been recently reported by Professor J. D. Blake.[1] F—— was found sitting by the roadside asking to be taken home. With slight assistance he was helped into a waggon, but upon trying to leave it three-quarters of an hour afterwards, he was unable to do so. He spoke, swallowed, requested water during the night, and asked for something to eat. In the morning of the next day he became stiff, his tongue was thick, he seemed stupid and drowsy. On the third day he was seen by Dr. Blake, who found him paralysed on the right side, and removed a spike driven through the skull into the brain. The spike was three and one-eighth inches long, and three-eighths of an inch in diameter. It was driven in till the head of the spike pressed firmly upon the scalp. Following its withdrawal was a small quantity of offensive decomposed blood and serum. After the removal of the spike there was right complete hemiplegia, sensation nearly abolished on same side. Cannot talk but understands, tongue runs to left side, answers questions by nods of the head, respiration slow, pulse full 94, temperature 103⅗°. On fourth day, pulse 98, temperature 104°, quiet, suffers no pain, swallows badly, slight muscular twitchings of the left side. On fifth day, profound stupor, takes nothing, pulse 98, temperature 104°, had convulsions, respirations 22, temperature in the evening 103⅗°, seems to be sinking. On sixth day, respirations 51, pulse very rapid, became convulsed, comatose, and died. Upon post-mortem it was found that the spike entered the skull through the left parietal bone near the median line, and one and a half inches from its junction with the frontal, passing obliquely downwards and forward, entered the left hemisphere just anterior to the Rolandic fissure, to open into it all the way down to the corpus callosum. The spike entered the lateral ventricle, the point slightly entering the intraventricular portion of the corpus striatum. In this case there are four causes of high temperature: meningitis, putrid blood, striate injury, and lesions of the convolutions about the fissure of Rolando. It is not likely that putrid blood or meningitis would cause so great an elevation (104°), and the lesion of the corpus striatum was not extensive enough. There is every reason to believe that the Rolandic convolutions bore the greater part of the injury, and were the cause of the elevated temperature. The case previously related had a temperature of 104°. Bourneville reports a case of softening in the neighbourhood of the fissure of Rolando with a temperature of 105°.

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[1] 'Guy's Hospital Reports,' 1884.

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[1] 'Philadelphia Medical Times,' July 1888.

Sylvian Centre.—In the cortex of cats there have been discovered the cuneate and Sylvian thermotaxic centres. Through the kindness of Dr. C. K. Mills, I have learned of a case which strongly supports the view of a thermotaxic centre in man about where I discovered the Sylvian. In the London 'Lancet,' Jan. 2, 1887, Dr. Page reported a case of depressed fracture of the skull, which was about the posterior part of the tempero-sphenoidal lobe, where a temp. of 105° F. existed. No other symptoms were present, and the patient was trephined on account of the high temperature. The operation caused the temperature to fall to nearly normal, not to rise again. The patient recovered.

Lesions of the Corpus Striatum.—Bourneville relates a case of a large patch of red softening in the left centrum ovale, and a recent patch in the right corpus striatum, where the temperature was 104·8° before death. Dr. White reports a case which undoubtedly shows the corpus striatum to be the seat of a thermic centre. W. C., æt. 6½, was admitted to the hospital. Before admission, he was paralysed on the right side, which came on suddenly, and was followed by incontinence of urine. On admission three months afterwards, he was found to be weak on the right side, never spoke, took no notice of things going on around him. For a month his temperature remained abnormal; but after that time had elapsed, the temperature rose to 102·4°, and the boy had a fit at the same time. About three weeks before his death he became completely unconscious, and remained so. Upon post-mortem no evidences were found of tubercle, meningitis, tumour, or disease of the cerebral arteries. Just above the anterior commissure of the brain was a brownish patch about a quarter of an inch in diameter, so soft that it left a hole when the brain was cut. These patches were symmetrical both in colour, size, and position; well-marked grey discolouration was present in both crura cerebri at their anterior inner margin. This degeneration extended down the cord to the lumbar region. All the other organs in the body were perfectly healthy. The table gives the temperature of this remarkable case.

March 24 to April 3	Temp. range 95°—96°
April 3 " " 17	" " 97·4°—98·4°
" 17 " " 24	" " 95°—97·4°
" 25	Morning 101·2°, Evening 104°
" 26	" 97·4°, " 99·2°
" 27	All day 97°
" 28	Morning 98°, Evening 100°
" 29 to May 25	" 96°—99°—100°
May 26	" 100·2°, Evening 106·6°
" 27	" 105°
" 28	Kept at 102° by sponging
" 29	Morning 102·4°, Evening 99·2°
" 30	" 105° (convulsions)
" 30 to June 5	102° by sponging
" 6 " " 8	98°—100°
" 8 " " 11	102°
" 11 " " 16	103°
" 17 " " 18	105° Just before death 107°, fifth exacerbation. There was wasting of muscles all over the body.

A similar wasting of the muscles is seen in animals whose thermic area in the corpus striatum is injured.

Dr. Bagojawlensky[1] has reported a case of elevated temperature where the only lesion was echinococcus cysts in the corpora striata.

Thalamic Lesions.—The pathological cases in this area are not very much to the point. Ollivier reports a case of hemorrhage into the optic thalamus and crus cerebri with a temperature of 102°. Dr. Beevor reports a case of a boy, æt. 11. Present illness came on five months ago. Four months ago he had a fit, afterwards paralysis of the muscles of the eyes, left arm, left leg, and left face. On admission, all the above parts were paralysed. There was intense optic neuritis. He was deaf, and passed urine and fæces under him. Persistent priapism was present. The temperature the evening before death was 104°, just before death 107·4°. Upon post-mortem there was found an abnormal growth, three inches long and two and a half broad, starting from the upper surface of the cerebellum, extending forward between the optic thalami, flattening the corpora quadrigemina, pressing upon the right crus and the right superior cerebellar peduncle. This case lends some support, but not a very strong one, that the thalami are concerned in the elevation of temperature.

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[1] 'Centralblatt f. Med. Wiss.,' 1888.

Subnormal Temperatures.—It has been observed in many of my experiments upon animals that after an irritation of basal heat-centres the preceding elevated temperature was succeeded by a temperature and a heat-production below normal. Dr. Farnham has noted in a girl æt. 3 years with typhoid fever a similar state of affairs. Through the period of low range of the thermometer, beyond a slight irregularity of the heart's rhythm, the child developed no unfavourable symptoms, but gained steadily in strength, slept well, and ate in a satisfactory manner. The table shows the subnormal temperatures, taken with a Hicks' thermometer.

Up to November 9th, the temperature is rectal, afterward axillary.

	Morning.	Evening.
Nov. 7	104·8°	105·8°
„ 8	103·2	106·2
„ 9	102·2	104·2
„ 10	101·0	104·4
„ 11	—	103·4
„ 12	101·2	103·0
„ 13	99·6	102·8
„ 14	98·8	101·2
„ 15	97·6	100·2
„ 16	96·6	98·8
„ 17	95·4	97·4
„ 18	95·4	95·2
„ 19	96·0	97·0
„ 20	95·4	96·2
„ 21	95·6	97·2
„ 22	95·2	97·4
„ 23	96·6	97·8
„ 24	96·6	97·8
„ 25	97 0	98·2
„ 26	96·4	98·4

In consumption the temperature, according to Dr. Williams, is as follows:

I. A wide range of temperature extending from 94° F. to 103·4° F.

II. An afternoon pyrexia.

III. Subnormal temperature in the early morning, indicating the collapse influence.

Convulsive Affections.—In another place I have advanced reasons for believing that the high temperatures of tetanus are due to an excitation of the spinal thermogenic centres, and not to muscular movements. In an analysis of 410 cases of chorea, Dr. Osler arrived at the conclusion that fever takes place when rheumatism sets in, when endocarditis becomes severe, and in some cases of chorea insaniens. There may be the most intense and violent movements without any rise of temperature, and a great majority of cases remain afebrile throughout. Certainly muscular movements have little to do with elevated temperature in chorea. In epilepsy Lemoine has made two hundred observations of isolated attacks, and arrived at the conclusion that the temperature rises with the attack, but falls rapidly afterwards, so that even in a quarter of an hour it may be a little below normal, but after two hours it was about normal again. The average rise is 1·2° F., and the rectal temperature very rarely goes above 102° F.

Tumours and lesions of the spinal cord, oblong medulla or pons varolii, are all usually followed by high temperature. These cases are explained as being due to a removal of the influence of the thermotaxic centres, allowing spinal thermogenesis to become exaggerated. Low temperatures here are due to excitation of the thermotaxic centres, their fibres, or to affections of the thermolytic apparatus.

High Temperatures.—There have been reported a series of cases where no adequate cause was found, but considering the extensive apparatus subservient to temperature, the lesion has probably been overlooked. Teale's case of injury to the spine with a temperature of 125° and Mahomed's of 128° are remarkable temperatures. These cases, if true, and I am inclined to believe them possible, are somewhat parallelled by the experiments of Eulenburg and Landois upon the Rolandic centre, where they found a difference of 23° F. between the extremities of a dog. Hence it is quite possible that a similar high temperature might exist in man, after a disturbance of the Rolandic heat-centres. The rapid and high elevation of temperature often seen in malarial paroxysms indicate to my mind an action of the poison upon the thalamic centre, as it is the most sensitive heat-centre, gives the highest temperature, and the rise and fall correspond closely to the curve of a malarial paroxysm.

Diagnosis.—How far thermal observations will aid in the diagnosis of lesions of the brain is a difficult question to answer. If the other symptoms point to a lesion of the pons, certainly high temperatures will be an important aid. If the symptoms indicate a lesion about the corpus striatum, then high temperature with general muscular wasting will be corroborative.

These fragmentary observations show, in my opinion, that heat-centres undoubtedly exist in man. The task of establishing the localisation with all the required precision now devolves upon neuropathologists.

HALLUCINATIONS, AND THE SUBJECTIVE SENSATIONS OF THE SANE.[1]

BY D. HACK TUKE, M.D., F.R.C.P.

In the observations which follow I do not restrict my enquiry to the hallucinations of the insane. I include the subjective sensations of the sane.

Several cases, raising oft-debated but still undetermined questions in regard to hallucinations, have led me to think that they may be again profitably discussed.

Among those which arise are :—

1. Has any advance been made in regard to the nature and seat of hallucinations since the period when Brewster enunciated his famous statement, that *all* hallucinations involve the corresponding peripheral sense-organ?

2. More especially, does the discovery of sensory centres in the cerebral cortex disprove this position?

3. If lateral pressure on one eyeball so as to disturb the axes of the eyes causes double vision, is it a certain proof of the external presence of an apparent object? (Brewster's test.)

4. What are the tests of the retina being involved in visual hallucinations?

The case of Mrs. A., observed by Brewster, has become historical, and has reference to the third question. She had laboured under auditory hallucinations when he met her, and he thought it well to anticipate the occurrence of visual hallucinations, or as he terms them "spectral illusions," so as to put on her guard against taking them for realities. Now the test which he gave her was this. He told her "she might distinguish a genuine ghost existing externally, and seen as an external object, from one created by the mind, by merely pressing one eye (or straining them both) so as to see objects double; for in this case the external object or (supposed) apparition would invariably be doubled, while the impression on the retina created by the mind would remain single."[1]

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[1] Read at the Psychology Section of the Meeting of the British Medical Association, Glasgow, August 10, 1888.

On the fourth question, Huxley speaks almost as strongly as Brewster, when he is referring to the case of Mrs. A. and the spectral cat which she saw. Huxley says, "There can be no doubt that exactly those parts of her retina which would have been affected by the image of a cat, and those parts of her auditory organs which would have been set vibrating by her husband's voice, or the portions of the sensorium with which those organs of sense are connected, were thrown into a corresponding state of activity by some internal cause."[2] It is true that there is a saving clause in regard to the sensorium, but he evidently does not dissent from Brewster. As is well known, Mr. Huxley can speak from personal experience, which he has thus recorded. "I know not," he says, "if other persons are similarly troubled; but in reading books written by persons with whom I am acquainted, I am sometimes tormented by hearing the words pronounced in the exact way in which these persons would utter them, any trick or peculiarity of voice or gesture being also very accurately reproduced. And I suppose every one must have been startled, at times, by the extreme distinctness with which his thoughts have embodied themselves in apparent voices."[3]

It may be well for a moment to formulate the elementary principles of mental physiology in regard to sensory perceptions in health, taking vision as our illustration, and confining ourselves to the visual perception of external objects. For our present purpose, a very brief statement will suffice.

a. A distinct image is formed on the retina. Double vision occurs for a time when the convergence of the axes of the eyes in the object perceived is prevented.

β. In the sensorium a visual sensation is excited, the nature of which is still very imperfectly understood. The ganglia of which the sensorium is composed are still undetermined. Whether the corpora quadrigemina are directly concerned with the visual sense appears to be uncertain.

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[1] He adds that his test occurred to Mrs A.'s mind when the hallucinations returned, but that "she was too well acquainted with their nature to require any such evidence of their mental origin; and the state of agitation which generally accompanies them seems to have prevented her from making the experiment as a matter of curiosity."—*Letters on Natural Magic*, 1832, p. 39.

[2] 'Elements of Physiology,' p. 240.

[3] Op. cit., p. 11.

γ. The sensory impulse is felt in the cerebral cortex, probably the occipital lobe, and according to Ferrier, in the visual centre in the angular gyrus, the molecular changes in which cause perfect visualization in the domain of consciousness—sensory impulses radiating to motor centres which are in relation to the muscles of the eye.

δ. Such changes in the cerebral cells—the results of the stimulus of an external object—persist as organic memory, stored up in the cortical centres.

If we adopt Dr. Hughlings-Jackson's mode of expression, we should have to say that in order to have a perception, or as he prefers to call it a vivid image of an object, say a brick, there is first the peripheral impression upon the retina, then secondly impulses pass through the lowest, through the middle,[1] and up to the highest sensory centres; from the most organised to the least organised; a multiplication of energy-liberations upwards, and consequently strong excitation and wide irradiation in the higher sensory centres. The physical condition in these centres, correlative with the red colour of the brick, has now been reached; but in order that the mind may become cognisant of its shape or the relation of its several positions one to another, which is acquired by ocular movements, there must thirdly be currents passing from the highest sensory centres to the highest motor centres, and from these downwards through middle and lowest motor centres to the peripheral muscles, causing movements of the eye-balls. The "vivid image" or mental state thus formed during the physical condition induced in the highest sensory and motor centres is, according to Dr. Jackson, "projected," because the lower centres are called into action, and thus it *seems* to form a part of the external world.

Passing on to the revival of sensory impressions in idea, we may say that—

1. Excitement of the cerebral cells which have undergone

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[1] Sensorium?

these changes induces re-presentation of the original object in thought.

2. Such ideal revivification of the object induces the sensation, in however faint a degree, originally produced by the presentation of the external object.

3. What has been affirmed in regard to presentative and re-presentative sensations in visual perceptions, holds good of all the senses; and moreover the excitement of one centre tends to excite other sensory centres also, and, it must be added, sensori-motor action as well.

Now up to this point there is a fair consensus of opinion among psychologists, both in regard to (1) the physiology of sensory perception, and of (2) the ordinary healthy recalling in idea of sensations previously perceived, or of their combination in fresh forms; but a difference of opinion exists as to the extent to which the original sensory tracts are traversed —whether indeed there is any extension beyond the cortical centres.

As is well known, Herbert Spencer insists upon the identity in kind though not in degree of our primary sensations or presentative feelings and our ideas or re-presentative feeling: "The visual feelings are of all others the most easily reproduced in thought. The redness of a soldier's coat, the blue of the sky, the whiteness of the snow-covered ground, can be revived in consciousness at any moment with scarcely an effort, and with relatively great clearness. The glare of an electric light may be so vividly conceived as to produce something like a sense of dazzling."[1]

Or as Ferrier puts it, "The molecular thrill of present sensation extending from the peripheral organ of sense is in the ideal sensation revived; but," he adds (and this is the important point), "as a rule not so powerfully as to extend to the periphery."[2]

Professor Bain seems to believe (as Brewster did) that the whole sensory tract is traversed in simply recalling an object, distinctly, as, for example, the rose which we have seen.

In view of our existing knowledge of the functions of the cerebral cortex, there is no reason to adopt this opinion. The thought of an object already seen involves, as Dr. Jackson has maintained, the highest sensory and motor centres only, the reflex action being confined to these central links of the sensori-motor chain. "The central part only of the whole process which occurred in perception is done over again, and, the excitations being slight, the image arising is faint, and the lower centres not being engaged, it is feebly and indefinitely projected, and *seems* more part of ourselves."[1]

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[1] 'Principles of Psychology,' vol. i. p. 229.

[2] 'The Functions of the Brain,' 2nd edit, p. 428.

Next, as to the conditions attaching to *after-images* consequent upon impression received on the retina by luminous objects. We can try experiments at any time for ourselves in regard to these spectra, which we know to be retinal. For our present enquiry, the following facts are important.

Nothing is more certain than that (1) lateral pressure on the eye-ball or a multiplying lens fails to double them, although I have heard the contrary stated by some who have tried the experiment. Again, such spectra (2) always follow the movements of the eye. So long as the retina remains impressed with the image, the latter (3) obscures real objects when we open our eyes and look on a dark surface.

Here, then, we have reliable tests for ascertaining whether a visual hallucination involves the retina or not (Question 4).

The images of vivid dreams may cause a sense of "projection into space," and remain for some seconds after the dreamer awakes. A young lady, a relative of my own, has experienced this on three occasions. On one she saw her sister in a dream, and then awoke, when the figure remained distinctly visible for a short time at the foot of the bed; and on another occasion, a lady's maid rushed upon her in her dream: she awoke and saw the spectrum of the maid in the room.

Dr. Bastian informs me of similar experiences in his own case. It may be premised that it has several times occurred to him to awake, after going to bed, out of a short first sleep of about half an hour or less, with a feeling of bewilderment and forgetfulness as to locality, at the same time receiving an impression from a window opposite of its being very small and close to him: gradually, however, the visual impression enlarged and receded, till it was seen as when in a state of perfect consciousness; and as gradually too the feeling of doubt as to locality cleared up, and he became conscious of being in bed. It was on waking in such a condition as this (with face turned to wall) that he once imagined he was still sitting in his room reading, and that hearing a slight noise he turned round in his arm-chair, and looking towards the door saw a little fair-haired child standing with the handle of the door, just opened, in her hand. The child was the daughter of his landlady, and waking (perhaps from some noise) he seemed to see her so distinct and life-like for a space of several seconds, that a few seconds afterwards, on perfectly regaining consciousness, he was filled with astonishment to find it was all a vision, and that he was actually in bed. Notes of the occurrence were made in 1864, a few months after the circumstance happened. About twelve years afterwards he had a similarly distinct hallucination on waking out of a first short sleep. Then, as on the previous occasion, he had been for some time doing too much hard work. The figure he had seen in his sleep remained for some seconds after he awoke. In none of these instances were the tests just mentioned employed. The figure seemed however "projected," as in the case of the after-image of a luminous object.

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[1] 'Croonian Lectures," *Lancet*, 1884, p. 27.

I now proceed to mention several cases which bear upon the questions raised at the beginning of this paper.

1. A clergyman in Bethlem Hospital under Dr. Savage describes in a most graphic manner how he sees sentences addressed to him in legible type. He can distinguish the capital letters and see every word most distinctly. At first from his way of speaking, one supposes that he hears voices, but this is not the case. Thus he sees, not hears, himself addressed in the following terms: "I'll tell you where to go;" "I'm delighted with your misery;" "I'll stop up your passages." If he says what agony he is in, the words appear: "Yes, I'll give you worse;" "I'll make you break your head." "You must tear up all the Bibles and tracts." These sentences are continually communicated to him. One is, "Put up your spectacles, there's a good boy." The patient on telling me this last command, not unnaturally asks, "Can the brain do this?"

I have recently received the following short letter from him: "I do not think that I can add anything to what I have already said about my case, and can only therefore repeat that I am suffering from the never-ceasing presence in my head of *visible words,* and that, every moment. They are of a threatening character, such as 'I will give it you;' 'I have not done with you yet;' 'I will strike you with sores;' 'I know how to tease you;' and various other sentences too numerous to mention. Pray tell me whether you have heard of anything like this before? My agony is unceasing, and I cannot sleep." When his hat was one day blown off by the wind, he heard or rather saw the exultant words "I did it!"

This is the second attack, and has lasted five years. Twenty years ago, after having suffered in the same way for some years, he was operated upon by Sir H. Thompson for stone, and the result was instant recovery. The earliest symptom had been renal colic, followed by depression, and after a short time the appearance of words written in his brain. It should be added that Dr. Savage has requested Sir Henry Thompson to examine him during this second attack, but without any result beyond finding enlarged villi on the surface of the bladder. The instant disappearance of the visual hallucination after the operation would seem to show that, apart from the mental depression which might be referred to his gouty condition, the peripheral irritation of a calculus in the bladder acted on the cerebral centres and induced visual hallucinations. Did this act by inhibiting the highest mental level and allowing the lower level only to act? I conclude that in this patient's case the retina is not reached, for the words do not give the impression of being "projected," but seem to him depicted in his brain, and, as regards the effect of pressure on one eye-ball, no doubling of the words is caused, and he says they do not follow the eye.

In his case, then, the subjective image (1) is not projected, (2) is not doubled by pressure, (3) does not follow the movements of the eye, (4) does not cover objects, (5) is the same whether the eyes are open or shut.

2. The next case (H. J.) is that of a young man, also in

Bethlem Hospital, and is of special interest, because the visual hallucination of which he is the subject is a fixed one, a somewhat unusual circumstance. He has for a long time seen the figure of a man which he describes with great clearness, who is dressed in a blue coat, stands upon some scaffolding, having his left arm behind his back, while the right beckons the patient to him. Although he never speaks, the patient feels impelled to go in the direction the figure indicates by his hand, and this actually led the patient on one occasion to wander about till he was taken up by the police as a wandering lunatic, and it was on the same day I examined and certified him.

As this youth was quite willing to answer questions or try any experiment, a better case could not have presented itself, for trying the effect of altering the axes of the two eyes by pressing on one of them while the visual hallucination was distinctly in sight. Having first tested him with a picture hanging on the wall opposite, I found that under these conditions he at once said he saw two pictures instead of one. I got him to look steadily at the man in blue on the scaffolding, and, when he saw him before him as clearly as the picture on the wall, I pressed one eye in the same way as I had done before. Had he said he saw double, his reply would not have been conclusive; but as he said the figure remained single, I have no doubt that this was so, and that his case may be taken to show, so far as one case can do so, that subjective visual sensations are not doubled by interfering with the axis of vision. I repeated the experiment subsequently, with the same result. The hallucination became gradually fainter and fainter, and he ultimately recovered. It was interesting to watch the fading away of the image. He had ceased to believe in its reality long before it vanished. Here the image (1) was projected, (2) was not doubled by pressure. I have also no doubt, but this was more difficult to prove, it (3) followed the movements of the eye, and (4) was obscured by external objects.

3. The third case is that of a patient attending one of the London hospitals, about whose mental condition I was consulted some months ago. He complained of constantly seeing before

him the head of a dog. On examining him, I found that he was labouring under suspicious symptoms of general paralysis, but very intelligent in conversation, though losing his memory. Desirous to discover whether I could double the spectral object, I carefully tested him. He declared that when I pressed on one eye-ball he saw two dogs, but I was not convinced that he distinguished between the dog's head and the objects in the room, especially as he saw it distinctly only when he looked at the fire. On a subsequent occasion my doubts were confirmed. I cannot therefore allow this case to count as an exception to my experience in other cases. It is probably a case of illusion. An illusion may be doubled.

4. I pass on to a remarkable example of visual spectra in a perfectly sane person, upon which the above-mentioned experiment has been repeatedly tried, and with the result that no doubling the spectra took place.

Thanks to the courtesy of Dr. Lombard, of Norwood, the author of the well-known work on the 'Regional Temperature of the Head,' I am enabled to mention his own personal experience. Those who know his writings well know the conscientious accuracy which characterises them. The case is so conclusive and is so interesting on other grounds, that I must mention some of the particulars, especially the discovery of his singular power.

He had for some years occasionally seen images of persons and things which he had been attentively regarding, but he took little notice of the circumstance, and they speedily disappeared from the field of vision. On the 3rd of December, 1862, however, a much more decided visualization occurred. He was then in the army, and stationed on the west shore of Maryland. A military man, his daughter, a naval officer, and Dr. Lombard were seated in the verandah of a house which stood within a stone's throw of Chesapeake Bay. The girl sat on the second of the steps leading down from the verandah. He himself was on the same step at the other end, there being about five feet between them, and her profile being clearly in view. For no particular reason he began to stare at her, at the same time concentrating all his attention upon the features. At the expiration of about 20 minutes, he turned his eyes towards the bay simply to relieve them, when he saw before him the image of the girl very distinctly, and also in like manner all the objects that had been within the range of his vision. The image began almost at once to fade, but he found that by fixing his whole attention upon it that he could retain it. To this power I particularly draw attention. He next looked over her head towards a wood of Southern pine-trees, and again saw the image. He had repeated this experiment four or five times when the girl turned her head towards him, and asked why he was staring at her so. Dr. Lombard described what he had seen, when to his surprise she laughed and told him he was only a beginner, and that he ought to be able to obtain in 20 seconds what had taken as many minutes. She then led him on the verandah to a point where the moon shone brightly, and asked the naval officer to take note of the time occupied in the experiments. She told Dr. Lombard to keep everything but herself out of mind, to look her full in the face, and then to run his eyes up and down her figure. In a few seconds she called "Time!" and told him to look towards Chesapeake Bay, directing him to regard the image as attentively as he had regarded her previously. He then saw the image very distinctly. The time was 22 seconds. In the course of the same evening many more experiments were made. In reply to her father and Dr. Lombard, she said that she had discovered that she possessed this power through a schoolfellow two years before, who possessed the same faculty of voluntarily recalling spectral images. Dr. Lombard asked her whether this power would last any considerable length of time. She said she could only answer for two years from her own experience, upon which the naval officer spoke up, and said that he could answer for nearly fifty years, having exercised it himself since he was fourteen or fifteen years of age.

Here then were four instances,—Dr. Lombard, the naval officer, the young lady, and her schoolfellow—who could by intense mental attention recall figures which they had once succeeded in fixing on the mental tablet. I may say, that Dr. Lombard never sees faces other than those which he has known and regarded attentively.

Is there conclusive evidence that Dr. Lombard's "faces" involve the retina? One important test which was tried in his case—namely, whether the spectral figures moved with the movements of the eyes—yields an affirmative reply. If Dr. Lombard immediately after receiving the impression of a face brings up the image on the very spot, it will appear at nearly the same distance as the reality, and it will follow the motion of his eyes. This happened a short ago at Prof. B——'s house at Hanwell, where Dr. Lombard took (in photographic language) Dr., Mrs., and Miss X—— together, and each separately. At his own house in Norwood, at the present time Dr. Lombard sees them as if at a great distance, although perfectly distinct.

I enquired of Dr. Lombard whether the spectra concealed real objects in the room, or whether, on the contrary, he saw objects clearly through them. He says that he never sees a real object through a *good* image, but at times he has thought he could see more or less through an indistinct one. The concealment of external objects by ocular spectra is natural, and Brewster in commenting upon Mrs. A.'s case explains it by the recognised laws of optics. He held that this fact proves that the revival of an image does actually involve the retina. It is on record, however, that a gentleman who after waking from a dream saw the imaginary figure of which he had dreamt, particularly noticed that it made no difference in his seeing real objects behind the spectre. Can it be that when an object which has once been depicted on the retina is resuscitated as in Dr. Lombard's case, the image appears on the retina; but that when a purely fanciful picture, born of the fancy or of dreams, appears in spectral form, the cerebral visual centre, not the retina, may be the seat of the revived impression? Mr. Sully suggests to me that this might depend, not on the periphery being involved, but on the form and persistence of the image, which would make simultaneous attention to real objects impossible. Brewster's Mrs. A. saw the spectra of her husband and her cat, and they obscured her sight of objects in the room, but I am not able to say whether this occurred when the purely imaginary phantoms she describes appeared, nor can I say whether the visual hallucinations of the insane generally obscure real objects.

The power of transferring a single person or a group to another place, described by Dr. Lombard, is very remarkable He states that it requires a very strong concentration of the attention indeed.

Again, if two objects are very much alike, he can see their mental images together; but if he looks at one and tries to put the image of the other beside it, he will see only the reality. The other is invisible, if the resemblance be perfect, or it is a hazy object if there be some difference between the two.

I should say that the colours of the original objects are perfectly preserved in the spectra.

It must also be observed that Dr. Lombard never experiences any pain in his eyes after recalling spectral forms.

Before passing to another case of visual spectra, I would note that the revived images of Dr. Lombard (1) are projected, (2) are not doubled by pressure on the eye-ball, (3) follow the movements of the eye, and (4) cover external objects.

5. I have a young man under my care at the present time who labours under the delusion that he is dead. When I last saw him, he said he had distinctly seen the devil on the way. I asked him to draw the figure, which he immediately did, as you see here.[1] Roughly drawn as it is, it has evidently been done with conscientious accuracy. As he no longer saw the spectre I could not try any experiment upon the eyes. In his case the hallucination must have originated in the cortex, and not in the visual organ; just in the same way as an auditory hallucination must have had a cortical and not a sense-origin when a City man having lost his property becomes insane, and hears after a time a voice saying to him, "You are ruined." This is the sequence of events in insane hallucinations generally, but we cannot always retrace their history as in this instance.

6. A London physician called upon me just after this patient had drawn this figure,[1] and it elicited the interesting observation that both he and one of his boys occasionally perceived ocular spectra. They afforded good examples of what Galton

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[1] The drawings were exhibited at the Lecture.

has called "visualization." The boy sees in a lane at a particular spot the figure of a little old woman, and is so accustomed to it that he mentions it in a matter-of-fact way without believing it to be real. His father, when a child, used to see a spectral figure in his bedroom, and can now resuscitate it with great clearness, as he did for my benefit when he described it to me. The room came up before him, and the figure in one corner, which was not in the direct line of vision but on one side. The moment he turned his gaze to this corner it vanished. He could only see the image when he looked in the same direction as that in which he looked from his bed when a child. While he did so I got him to press on the eye-ball of one eye, but no doubling of the image happened. On the other point whether the spectrum followed the movements of the eye, he says it does so. In his case, then, the image is (1) projected, (2) is not doubled by ocular pressure, (3) follows the motion of the eyes, and (4) covers external objects.

7. Of visual hallucinations in another person of perfectly sound mind, I had a very good example in a late medical friend, who was not in the least troubled with his spectral visitants. At my request he sketched some of them, and being a skilful draughtsman he made some striking portraits, which show that as in Blake's case he saw them as distinctly as if they had been actually present. [Drawings shown.] Unfortunately no experiments were made as to pressing the eye-ball, or observing whether the figures moved with the movements of the eye, &c.[1]

As I have, in the foregoing cases, given my own experience as to the non-doubling of subjective visual images by disturbing the axis of vision, it is only right to state that Despine of Marseilles records the case of an insane patient who had a visual hallucination of the Virgin Mary. He tried the effect of pressure on the globe of one eye, with the result, he says, that she saw two Virgins.

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[1] I have thought that a few illustrative cases would be more instructive than the large number it would be easy to bring forward, but which would obscure the salient points I wish to keep clearly in view. I hope, however, at a future time to utilize the examples of hallucination of which I have notes, and which I shall continue to collect.

Again, Professor Ball of Paris obtained the same result by a like manipulation in a female lunatic who saw visions.[1]

Lastly, experiments by Binet and Féré in Paris on hypnotised subjects may be cited in support of the same experience. Time will not allow of my quoting these interesting experiments, but it is impossible to ignore them in any study of the nature and seat of hallucinations. Great credit is due to these observers for the extremely ingenious experiments referred to, and for the care they have evidently taken to avoid sources of fallacy. Whether, in so difficult an enquiry, they have always succeeded may, perhaps, be questioned; but I shall assume that the facts are exactly as they are described. It is only necessary to recall one instance. During hypnosis, a portrait is, by the suggestion of the experimenter, made to appear on a piece of cardboard. A visual hallucination is thus excited, and, moreover, it is located in a particular spot. Now it is this element in the experiment—the localisation of the hallucination on an external surface—which renders the results arrived at inconclusive in their bearing upon the point now under consideration, although important from other points of view. The observers found that every test which proves an object to be actually perceived applied to the hallucination which they had induced. A screen, or closing the eyes, rendered the hallucination invisible, and it was doubled by lateral pressure on one eye-ball, or by a prism. It could be seen reflected in a mirror, and the use of a spy-glass was accompanied by precisely the same effect as when applied to any object in the room. The glass had to be focussed for the imaginary as well as the real object.

Here, then, hallucination and actual perception appear to be proved to be identical. The result seems altogether opposed to what has been maintained in this article.

At first I was at a loss to reconcile this result with my own experience outside the phenomena of hypnotism. But further consideration satisfied me that the exception is more apparent than real. It is not denied, it is expressly stated, that the essential conditions of the success of this experiment were: an external object actually in sight, and one occupying the same position as the induced hallucination. One circumstance alone suffices to indicate the presence of conditions which remove the phenomenon from the range of ordinary hallucinations. When the card upon which the imaginary figure was depicted was turned round the "hallucination" disappeared; if inverted, the fictitious portrait was inverted also. Hence it is evident that the *point de repère*, as Binet terms it, and the suggested hallucination have become so completely fused and associated in the subject's mind by suggestion that they are inseparable. What the hypnotised subject really sees doubled by lateral pressure, what is suppressed by closing the eyes, and so on, *is* an external object, and therefore subject to the conditions attaching to all real objects within the range of vision. On reflection, therefore, I do not find in the interesting and important experiments of MM. Féré and Binet any exception to the results arrived at on other grounds in this paper. And, if I understand M. Binet correctly, I think his position scarcely differs from mine. The explanation of the apparent discrepancy is to be found in the surmise I made in the case of the patient who saw a dog's head when he looked into the fire. It is an *illusion*, not a hallucination—a change wrought in the appearance of an object by the fancy. It need hardly be said that an illusion would obey the same laws as an ordinary object of perception. In connection with this aspect of the question, it is necessary to find out, what seems to have escaped observation, that most of those who have referred to the Brewster test of objectivity have made a singular mistake in regard to it. In fact, they have completely reversed his formula. As already fully stated, his test was intended to distinguish between objective and subjective visual sensations. In the former case, the image would be doubled by lateral ocular pressure; in the latter, not. Remove the external stimulus, and doubling the image is no longer possible. So said Brewster; but the writers I refer to represent him as holding that mechanical pressure of the eye doubles the retinal image induced by central excitation—a hallucination. Thus M. Binet writes: "Brewster performed this experiment on a subject who had visual hallucinations, and pressure on the eye caused a duplication of the imaginary object."[1] Brewster's intention, on the contrary, was to prove by non-duplication that the figure seen was a hallucination, and not a ghost. Again, I am surprised to find M. Déspine writing: "Brewster noticed that, in destroying the parallelism of the two eyes during a hallucination of sight, by pressure exercised on the external side of one of them, the patient upon whom he experimented saw the object of his hallucination double, exactly as we see a real object when we squint."[2] Déspine then mentions the already-referred-to case of his own in point, and it must count as one apparently opposed to my own experience. But what I am concerned to show now is that Brewster would have declared that the Virgin which M. Déspine's patient saw was real instead of being a fancy, from the very fact that the figure was doubled. It is still more surprising that so acute a mind as Mr. Gurney's should have misunderstood a British writer's argument on this subject. "The opposite party" (to Esquirol) "pointed out," he says, "that visual hallucinations often vanished when the eyes are closed; or, as Brewster first observed, that they may be doubled by pressing one eyeball."[3] Unfortunately, the false impression Mr. Gurney received from the study of Brewster, invalidates, to a certain extent, his treatment of the nature of visual hallucinations. In short, he, like many others, entirely confounded the tests of whether or not an apparent object is actually external to the observer, and whether the retina, as well as the visual centre, is involved in a given case of hallucination of sight.

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[1] 'Leçons sur les Maladies Mentales,' p. 74. The case is complicated, however, by the patient being at the time in a state of artificial somnambulism. The explanation is probably the same as that suggested in those reported by Binet and Féré.

It is difficult to conceive how the fact can be otherwise than Brewster stated, seeing that the reason why one external object is doubled by the proceeding in question is so obviously due to preventing the simultaneous action of corresponding points of the retinæ of the two eyes. The evidence ought, therefore, to be overwhelmingly strong in proof of the doubling of subjective images.

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[1] 'Annales Médico-Psychologiques,' Nov. 1881.

[2] 'Animal Magnetism,' International series, 1887, p. 227. As a matter of fact, Brewster only advised Mrs. A. to try this experiment, but she omitted to do so.

[3] 'Mind,' April, 1885, p 168.

I demur to the axiom which has been laid down, that an imaginary object is perceived under the same conditions as a real one. I believe they differ in at least three particulars. First, in the effect produced by ocular pressure; secondly, in the behaviour of the image during movements of the eye; and thirdly, in the result of interposing an opaque screen between the observer and the object alleged to be seen.

I should substitute for this axiom the following: An imaginary object can be perceived under the same conditions as after-images. When this happens, the retina is involved. It would be extremely interesting to know whether, in cases of optic nerve atrophy associated with hallucinations, any tests have been employed to ascertain whether the conditions present were the same as those observed when the retina is known to be the seat of after-images.

I proceed to state Brewster's position in regard to the revival of sensory impressions in idea, and also visual hallucinations (Question 1).

"I propose," he says, "to show that the 'mind's eye' is actually the body's eye, and that the retina is the common tablet on which both classes of impressions are painted, and by means of which they receive their visual existence according to the same optical laws. Nor is this true merely in the case of spectral illusions; it holds good of all ideas recalled by the memory or created by the imagination, and may be regarded as a fundamental law in the science of pneumatology. . . . Spectres conjured up by the memory or the fancy have always a 'local habitation,' and appear in front of the eye and partake of its movements exactly like the impressions of luminous objects after the objects themselves are withdrawn." Thus Brewster asserts that not only spectral illusions (hallucinations), but ordinary mental pictures or ideas, are impressions on the retina, differing only in the degree of vividness with which they are seen, "being rendered more brilliant by some accidental and temporary derangement of the vital functions."[1]

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[1] 'Letters on Natural Magic,' p. 49.

Dismissing Brewster's opinion so far as it relates to ordinary mental picturing of objects in idea, and confining myself to his dictum in regard to visual hallucinations, or, as he calls them, "ocular illusions," I would place in direct antithesis to it that of Esquirol, with whom Falret, Lélut, and other high French authorities concurred. Now Esquirol asserted that "Hallucination is a cerebral or a mental phenomenon, produced independently of the senses." . . . "One can only conceive the existence of this symptom by supposing the brain to be excited by a certain cause. . . . The images, ideas, notions, which appear to form part of the functional change in the senses present themselves to the mind with the same characters, and have the same seat, namely, the brain."[1] In short, according to Esquirol, the sensory nerves and peripheral sense-organs have not their functions called into requisition at all.

Well, if this is too unqualified a statement, as no doubt it is, for surely some hallucinations are due to peripheral pathological changes, it seems to me to be nearer the truth than the opposite extreme advocated by Brewster. For how can it be universally true when hallucinations of a special sense occur in the absence of the organ? Thus, visual spectra or hallucinations, may arise when the optic nerves are atrophied. Esquirol records the case of a Jewess who, although totally blind, laboured under extraordinary hallucinations of sight. She died, and he found that both optic nerves were atrophied from the point of their decussation to that of entering the eyes. In this instance, as he observes, the transmission of visual impressions to the retina was impossible.

Another patient under Esquirol had visual hallucinations, and became blind for seven years. Did this circumstance made any difference? Not the slightest. The hallucinations continued in full force all that time. He died, and Esquirol found the optic nerves of a grey colour, in fact, like wet parchment; flattened and atrophied throughout their whole course.[2]

These are merely illustrations of the instances in which sense-impressions have been capable of revival, although the peripheral sense-organs have not been able to perform their functions. We are therefore driven to conclude that it is not absolutely necessary in even vivid hallucinations that the latter should be involved in their production. Stored up sensory images in the cortical centres may suffice (with or without the participation of the sensorium) for this revival. The increased knowledge we possess of the former supports this view.

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[1] 'Maladies Mentales,' vol. i. p. 200.

[2] Op. cit. vol. i. pp. 187–8.

There must, however, be sensory images to recall. On enquiry of Mr. Buckle, B.A., the Superintendent of the Wilberforce School for the Blind at York, who kindly investigated the subject, he informed me that those pupils who have no remembrance of having ever seen, never had had dreams of seeing objects, or experienced visual hallucinations. On the contrary, they were inclined to doubt the veracity of any blind persons who asserted that they had experienced them. Mr. Buckle found that some of the pupils who dreamed of domestic animals, dreamed of them as talking like Æsop's characters. I may add, that hallucinations of touch are by no means uncommon, pupils stating that their hands feel as if they were holding a ball or apple.

Here must be noted the familiar illustration of the patient who, although his leg is amputated, has vivid sensations of pain in his toes. No one doubts that the brain (sensorium or cortical sensory centre) may be the seat of a sensation which is mistakenly referred to a peripheral nerve-ending or sensory organ which no longer exists.

Whether, as a general rule, the peripheral terminations of the sensory nerves are reached in hallucinations is, however, another question.

The ordinary view is that promulgated by Carpenter, namely, that there is a backward current through "the nerves of the internal senses" to the sensorium, exciting sensations there precisely identical with those experienced when external objects are perceived, and the sensorium is reached through the nerves of the external senses.

According to Dr. Hughlings-Jackson, when there is distinct subjective perception of an object as in vivid hallucination, there is a stronger discharge of the same nervous arrangements of the highest centres as in actual perception of an external object, so that the middle and then the lowest centres are overcome.[1] From his standpoint, it does not follow that because a patient labours under a hallucination, that that particular part of the cortical centre concerned in the special sense involved is diseased. Visual perception, and retinal impressions are represented in the highest cerebral centres, and when we recall to memory an object, there is not only the "purely psychical state," but a correlative physical state, namely a discharge of some nervous arrangement of our highest centres representing parts of the body, including impressions on the retina. Dr. Jackson's well-known theory of nervous discharges in epileptic fits accounts for the visual or auditory hallucinations which occur at the commencement of some of these attacks, as being mental states which "arise during slightly raised discharges of healthy arrangements untouched by the epileptic discharge.[2]

Discharging lesions of the cerebral cortex in epileptic attacks when accompanied by visual auditory or other aura, constituting one form of hallucination, bear so far upon this theory that they point to the origin of some hallucinations in the cerebral cortex and therefore disprove Ritti's position, based on the cerebral physiology of Luys, that all hallucinations originate in the basal sensory ganglia. It does not, however, follow that the impulse does not reach the peripheral sense-organs. I will briefly refer to an unpublished case of a female patient at the National Hospital for the paralysed and epileptic, under Dr. Gowers, as an illustration of the foregoing, kindly placed at my disposal, by Dr. Wilson of Wolverhampton who made the autopsy.

Six weeks before her admission, and three weeks after her confinement, she was standing and looking out of the window, when she suddenly heard a dreadful noise "like the clicking of machinery." She fell, losing consciousness and becoming convulsed. A fortnight afterwards, she had five similar attacks. The patient had a history of syphilitic sores, rash, and sore throat. For this she was treated by Dr. Gowers, and improved. After leaving the hospital, however, the fits returned, accompanied by the same auditory hallucinations as before. There were no more seizures, but she died not long after, conscious to the last.

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[1] "The Croonian Lectures," 'Lancet,' 1884, p. 24.

[2] Loc. cit.

The autopsy revealed a tumour in the right superior temporo-sphenoidal convolution, about one and a half inches in diameter. For my present purpose, there is no need to describe the tumour or its surroundings in detail further than to add that it involved the middle temporo-sphenoidal convolution. The basal ganglia were quite healthy.

My attention was directed to this case by Professor Horsley, who, I may mention, not only holds that the cortical sensory centres would amply suffice for all sensorial hallucinations, however vivid, but hesitates to admit that there is any certain proof that in hallucinations, a current or impulse ever passes backwards down a sensory channel to the sentient nerve ending.

A case of epilepsy under Dr. Hughes-Bennett's care may also be mentioned. The patient had received a severe blow over the region of the angular gyrus some years before. A bright red flash of light usually preceded the fit. This was followed by visual hallucinations of different objects, judging from the manner in which he acted and spoke. These symptoms disappeared after an operation performed by Mr. Pearce Gould at the spot indicated by an old cicatrix. In his usual state the patient enjoyed normal vision.[1]

The reference to Dr. Hughlings-Jackson's views already made will indicate the mode in which he would explain the occurrence of hallucination in these cases.

It may be urged in favour of the Brewster doctrine that those patients who suffer from auditory hallucinations stop their ears with tobacco, etc.

It so happens that my attention was first definitely directed to the subject of hallucinations at the York Retreat, where my room looked into an airing court in which a male patient stood, day after day, carefully stopping his ears with his fingers. I was aware that he had auditory hallucinations, but it seemed to me extraordinary that as these were of subjective origin he should gain any relief from closing the outward ear

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[1] 'British Medical Journal,' Jan. 1, 1887.

From that time I have endeavoured to ascertain from patients who hear voices whether any such relief is afforded by this proceeding, or again whether those who labour under visual hallucinations derive advantage from shutting their eyes.

It is indeed possible that no more relief is derived from the actions of patients in this respect, than can be accounted for by their naturally imagining that such relief can be obtained by shutting out what they regard as external influences and agents. There are cases, however, in which a local affection of the peripheral termination of the sensory nerve is the point of departure of the hallucination, and in such instances pressure on the ear, for example, by the fingers may afford some temporary comfort.

Again, in the case of auditory hallucinations of unquestionable cerebral origin, there was pain felt "in the ears." The patient was fresh from a three days' revival in London, under the auspices of the Established Church, and was brought to me hearing voices sending her to hell, along with distinct pain, as I have said, in the ears.

In Sir David Brewster's case (Mrs. A.), mention is likewise made of an uncomfortable sensation felt in the eyes when she saw phantoms. Again, it is so mentioned in Dr. Lombard's case.

Lastly, in some experiments in Hypnotism in Paris, it is stated that when visual hallucinations were induced, the subject complained of pain in the eyes. I do not wish to put any of these instances out of court; I only wish to point out how easily a person may refer to the periphery, through association of ideas with certain parts, that which does not extend further than the sensory centres.

Indeed, the fact that the general sensitiveness of the eye may be increased in the hypnotic subject of a visual hallucination, just as happens when a real object is waved before the subject's eyes, has been pointed to by M. Féré as a proof "that visual hallucination excites the visual centres."[1] He suggests that there are sensory centres common to the organs of the senses and to the integuments which cover them.

On the whole, then, I should say this of Brewster's position:

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[1] 'Animal Magnetism,' p. 259.

that although it no doubt holds good in many instances, it is certainly not essential to the explanation of all hallucinations. As respects Esquirol's position, while it goes quite too far in ignoring the occasional origin of hallucinations in the sense-organs themselves, it seizes the important truth of the affection of the brain itself in hallucinations. Still, this great alienist could not bring to the support of his doctrine the knowledge we now possess of the functions of the cerebral cortex, and in answering the question, Does the discovery of sensory centres in the cortex disprove the position that all hallucinations of the senses involve the peripheral sense-centres? I should reply that it decidedly helps us to deny that such is absolutely necessary.

There is another theory of the pathogeny of hallucinations now in favour in France, based by Ritti upon the cerebral physiology of Luys which differs from that of Esquirol as also Brewster, and teaches that all peripheral impressions are condensed in the grey substance of the optic thalami, whence they irradiate towards the various regions of the cerebral cortex, and that in hallucinations there is a spontaneous activity of these ganglia with the like result, the cortical cells acting on the erroneous indications conveyed to them as if they were real. This theory, which is adopted by M. Christian, the distinguished head of Charenton, in the recently published French Encyclopædia of the Sciences,[1] does not appear to me to be satisfactory, for this reason among others, that it ignores that large class of cases which unquestionably originate in what we call mental, that is, cortical changes.

The difficulty of deciding whether, in hallucinations, the terminal sense-organs are involved, as well as the cerebral centres, confronts us also in the study of those hallucinations which are caused by the administration of drugs.

Dr. Lauder Brunton, who informs me that what follows correctly expresses his views, experimented on himself with a grain of digitalin, taken in the course of forty-eight hours, and found that it produced distinct subjective sensations of sight.

As a similar effect is produced by the local application of this drug, it would be probably safe to conclude that the periphery, as well as the centre, is involved when digitalin is administered internally. The visions induced by Cannabis Indica are well known. Here, on the other hand, it is probable that the cerebral centre is the seat of these visual hallucinations, because the higher mental functions are so obviously under the influence of this drug.

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[1] 'Dict. Encyclopédie des Sciences Médicales,' 1885, an able article by Dr. Christian of Charenton.

Again, auditory hallucinations produced by the administration of drugs may be supposed to have their seat, in some instances, in the cerebral centre of hearing, and in others in the terminal apparatus. As strychnia has the effect of intensifying the sense of hearing by its action on the brain, it would be interesting to know whether those auditory hallucinations which arise in insanity are rendered more vivid by the administration of strychnia.

Lauder Brunton is himself by no means sure whether the singing in the ears caused by full doses of quinine or salicylate of sodium is due to its stimulating action on the auditory nerve or the cerebral auditory centre, although, seeing that delirium follows the administration of still larger doses, the probability is that this singing does not involve the auditory apparatus, but the cortical centre. The action of quinine upon the cerebral centres seems confirmed by a case recorded by Dr. Brunton, in which a dose of quinine was followed by an epileptic convulsion.[1]

Again, as some patients complain of bad smells of clearly cerebral origin, it would be worth while experimenting with strychnia, because it has been found to have the effect in healthy persons of neutralising the character of disagreeably smelling substances, such as assafœtida, and rendering them even agreeable. How strychnia acts may be uncertain, but Dr. L. Brunton is disposed to attribute its effect not only to the action which is produced on the cerebral olfactory centre, but possibly also upon the olfactory nerve itself, inasmuch as peripheral action has been demonstrated in the case of the retina of the frog by recent experiments.[2]

These facts, few though they be, lead me to hope that the further study of the influence of certain drugs upon the nervous system in inducing subjective sensory phenomena will throw some light upon the pathology of the hallucinations of the insane.

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[1] 'Pharmacology, Therapeutics, and Materia Medica,' 3rd Ed., 1887, p. 190.

[2] This point is recorded and illustrated in Dr. Brunton's work, 'Op. cit.' p. 227.

Do we gain any light from the occurrence of unilateral hallucinations? Are they induced by peripheral changes, or are they of central origin? Dr. Alexander Robertson, in a paper read at the Psychology Section of the International Congress, 1881, explains unilateral hallucinations by supposing that there is an original difference in the sensory centres of the cerebral cortex; one, in fact, being feebler than the other. He applies Sir James Paget's views on general pathology: namely, that "when a part is congenitally weaker than it should be, or has been enfeebled by disease, though it may be restored to an apparently healthy state, it is apt to be affected first, and to suffer most when a general morbid action arises in the system." Thus, alcohol might cause unilateral hallucinations, according to Dr. Robertson, by acting on the feebler cortical sense centre of one side.

On the other hand, M. Régis has endeavoured to show that unilateral hallucinations are an unanswerable proof of the intervention of the sense-organs themselves in hallucinations, the lesion in those hallucinations which are unilateral arising in the periphery, and being propagated gradually to the sensorium or cortical centre.

He adduces several interesting cases. In one, a patient heard voices applyi[illegible]thets to him, in only the right ear. He la[illegible]orrhœa of this ear for a [illegible]y Pick of Prague) [illegible] the left eye, and

The cases no doubt disprove the exclusive doctrine of Esquirol, that the sense-organs are in no wise concerned either primarily or secondarily with hallucinations; but they do not militate against Dr. Robertson's view, which would none the less apply to hallucinations not demonstrably of peripheral origin. Nor do M. Régis' cases touch the doctrine of Brewster, that centrally induced hallucinations must involve the sense-organs as well as the brain. I have already adduced evidence to show that Brewster's doctrine is not tenable in this exclusive way of putting it, any more than Esquirol's. No one can doubt that some hallucinations are of peripheral origin.

As an illustration of the uncertainty which surrounds this inquiry, and as a justification of introducing it for discussion, I may mention the fact that Dr. Alexander Robertson expresses himself very cautiously in regard to the sensory centres in the cortex in their relation to hallucinations. He observes: "I, to some extent, assume that the supreme centre in the cortical substance is the one involved. The fact that hallucinations are associated with other mental disturbances, and, indeed, in almost all the patients, with complete insanity, seems to point pretty distinctly to that as the correct conclusion. Still," he adds, "in certain cases disordered action may be limited to the sensorium." An interesting case in point is given, but Dr. Robertson makes the guarded commentary, "on the other hand, it is quite conceivable that the highest visual centres—the psycho-sensorial—may alone have been involved, just as the convulsive seizures li[illegible] the arm or leg disease may apparently be confi[illegible]ntre in the convolutions which [illegible] of the one or the [illegible] with the motions of the eye, and is "projected" when the observer looks on a dark ground.

3. If visual hallucinations present the same phenomena as those observed in these luminous after-images, they may be regarded as involving the retina.

4. If, on the contrary, these phenomena are not present, visual hallucinations do not involve the retina, being confined to the cortical centres, or possibly extending to the sensorium, and may occur when the optic nerves are atrophied.

5. The discovery of cortical sensory centres appears to throw light upon cases of hallucination not of peripheral origin, whether their abnormal action be regarded as primary, or as secondary to that of higher cortical centres.

ON A CONNECTION BETWEEN EPILEPSY AND ERRORS OF OCULAR REFRACTION.

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AND

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SOME years ago, when conducting an investigation into the condition of the fundus oculi in insane individuals, we tested the refraction of a considerable number of Asylum patients: a fair proportion of these were epileptics; and in the course of this enquiry we were struck with the large number of refractive errors which we met with in the epileptic patients submitted to examination—a proportion considerably greater than that obtaining amongst the other classes of patients.

The number of cases then examined was not, however, sufficiently great to render it safe to draw any general conclusions, and we were unable at the time to pursue the subject further.

Since then, however, one of us has paid special attention to the matter in the case of patients with simple epilepsy coming under notice in private practice; and last winter we conjointly took an opportunity of examining upwards of one hundred Asylum epileptics, and testing their refraction with as much accuracy as possible. The result of the investigation of the matter on these lines has been to confirm our previous idea with reference to the large percentage of abnormalities of refraction amongst epileptics, and to make it appear probable to us that in certain cases the refractive error and the epilepsy may stand to each other in the relation of cause and effect.

To prevent any possible misconception, we wish at the outset to make clear that we have no notion of suggesting that all cases of epilepsy have their origin in ocular defects, but only that a certain proportion, at present undetermined, are thus produced. Nor indeed do we think that refractive errors, however great, would be capable of producing this affection in the case of individuals with thoroughly stable brains. But, given a predisposition to epilepsy, we believe that an abnormal condition of the refractive media of the eye may, at times, prove the exciting cause of the disease.

We are indeed now familiar with the fact that nervous troubles of other kinds have not unfrequently such a starting point. Headaches and neuralgias of different sorts frequently owe their origin to ocular troubles, and disappear when the patient is supplied with accurately adjusted glasses. There seems, therefore, nothing unreasonable in the idea that epilepsy may at times be similarly produced. Our aim in the present communication is simply to state what facts we possess in support of this assumption.

In our former enquiry we examined forty-eight cases of epilepsy associated with insanity, and we found that "out of the forty-eight cases, no fewer than twenty presented errors of refraction; of these twelve were hypermetropic, and two had hypermetropic astigmatism, two were myopic, and the other four had myopic astigmatism."[1]

Our present investigation may be divided into two parts. The first deals with a series of one hundred and three unselected cases of epilepsy associated with insanity, the patients being of both sexes and all of them inmates of Rainhill Asylum. The second division comprises nine cases of simple epilepsy which have come under notice in private practice.

We will consider the former class first. The cases coming under this head were taken altogether without selection, the object of the investigation being to determine the proportion per cent. of refractive errors in the class examined.

All the cases exhibited more or less mental defect associated with the epilepsy, some of the patients being imbeciles, whilst others had acquired their insanity later in life; but in every case, so far as known, the epilepsy had been the primary thing, and the mental trouble secondary. In all one hundred and three cases were examined, of which sixty-one were males, and forty-two females. Of these one hundred and three cases, we considered fifty-five to be emmetropic, including under this head such cases as did not manifest a higher degree of hypermetropia than one dioptre; whilst forty-eight cases (46·60 per cent.) presented refractive errors of a more or less decided character. These were proportioned as follows:—Hypermetropia accounted for eighteen cases, and hypermetropic astigmatism (simple or compound) for seventeen more; three were examples of simple myopia, and in seven others the myopia was associated with astigmatism; of the remaining three cases, two came under the head of mixed astigmatism, whilst the third presented myopic astigmatism in one eye, and hypermetropic astigmatism in the other (anisometropia)[1]

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[1] 'BRAIN,' Vol. VII. p. 29 (April 1884).

A few words are desirable as regards the mental condition of the patients. Of the one hundred and three cases, twenty-six were imbeciles, that is, the mental defect was either congenital or had come on in early life; in all of these cases in which a history was obtainable, the mental dwarfing appeared to have been the result of the epilepsy, and it is probable that this connection obtained in all instances. Most of the other seventy-seven cases presented more or less dementia, and in not a few this was very decided, the patients being perfectly fatuous; thirty-nine of the seventy-seven might be put down as decided dements. The remaining thirty-eight also exhibited as a rule more or less mental failure, their condition varying according to the frequency of their fits; in most of these, however, the symptoms of mania or melancholia (chiefly the former) largely predominated over those of mental failure, and in some few cases the patients in the intervals between their fits were in all respects rational.

The percentage of refractive errors above given seems to us considerably higher than that obtaining in the population at large; but this does not in itself prove that the fits were in any case dependent on the ocular defects. To establish this point, it would be necessary to show that the epilepsy disappeared when the abnormality of refraction was corrected with glasses. From the above record it will, however, readily be surmised that these patients were very unfavourable subjects for the application of any form of treatment. So readily does the nervous system fall into a habit, that if it has been accustomed to discharge itself irregularly for any length of time, this abnormal discharge will in most cases continue, even if the cause which originally excited it be withdrawn. To no disease is this statement more applicable than to epilepsy; and by the time this disease has produced sufficient mental failure to render it necessary to place a person under Asylum care, the case has usually lasted so long as to have passed altogether beyond the reach of cure. Nevertheless, we fitted up a few of the more promising of these patients with glasses, without, however, feeling in any way sanguine as to the effect of this method of treatment; and so far we have not obtained any decided results. The patients whose numbers are given as 2, 5, 13, 15, and 20 in the table, were thus supplied with correcting glasses. Of these, No. 20 died from an attack of status epilepticus not long after he had obtained his glasses, and before sufficient time had elapsed to test the result of this mode of treatment. Nos. 5 and 13 refused to wear their glasses. No. 2, who has suffered much from headaches, finds great relief from his glasses, and his first pair having been broken in a fight with a fellow-patient, he gave no peace until he was supplied with another one: his fits, however, appear to continue much as formerly; and the same remark applies to No. 15.[1] The results, as we shall see, in the case of private patients have been more satisfactory.

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[1] We append a table showing the kind and amount of refractive error in each of these forty-eight cases.

It may be objected to an enquiry on the above lines that ocular defects may be part and parcel of an abnormal physical constitution which expresses itself in the epileptic neurosis, and hence that we might expect to find a larger proportion of such defects in epileptic persons than in normally constituted individuals. But if this were the case, we should certainly anticipate a higher proportion of refractive errors in imbeciles than in ordinary insane persons, since the former class comprise those who have either been congenitally defective, or who have broken down in early life—in whom therefore the hereditary vice has been greatest. But so far as our figures go the contrary is the case. Thus out of twenty-six epileptic imbeciles, we find fifteen to be emmetropic, the remaining eleven exhibiting some form of refractive abnormality—a percentage of errors of 42·30; whilst out of seventy-seven cases of epilepsy combined with other forms of insanity, thirty-seven exhibited abnormalities, giving a percentage of 48·05: so that a smaller proportion of refractive errors was found amongst the imbeciles than in the other classes of patients. The figures are not, however, sufficiently large to permit of decided conclusions being drawn.

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[1] The epilepsy in all these cases has been of many years' duration.

In the second portion of our investigation are included all cases of simple epilepsy coming under notice in private practice. We would particularly call attention to the fact that none of these patients sought advice on account of any ocular troubles, but they were all sent to us by their medical attendants, to whom we had mentioned in conversation our ideas as to a possible connection between epilepsy and refractive abnormalities. It is noteworthy that of all the cases, nine in number, thus sent to us for examination, not one was emmetropic. We think it desirable to give a short epitome of every such case which has come under our notice.[1]

CASE I.—*Epilepsy.—Hypermetropia.—Cure.*

MISS J. D., æt. 11½ years, was brought on Oct. 6, 1887, with a note from her medical attendant, Dr. Henry Gorst of Huyton, stating that she had had frontal headache for some time, and had latterly had a series of convulsive seizures, which he considered to be of epileptic character. It was ascertained that the convulsive attacks, about forty in number, occurred on one day only, and on that occasion the patient was unconscious from about 9.30 A.M. until 10 P.M.

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[1] Except when otherwise stated, no treatment other than the correction of the refractive error by glasses was resorted to after the case came before us.

Examination showed—

Right eye . . Manifest hypermetropia 0·75 D = [illegible]
Left „ . . . „ „ 0·50 D = [illegible]

Oct. 10, 1887 (after atropisation for four days)—

Right eye . . . Total hypermetropia 2·50 D = [illegible]
Left „ . . . „ „ 2·50 D = [illegible]

Spectacles of +1·50 D were ordered for near work, but were not insisted on for distance unless she had another fit. On May 2, 1888, the patient's mother wrote to say that the child "has had no more fits, and is free from the constant headache she suffered from. She now wears her glasses constantly." On further enquiry under date Aug. 21, 1888, the reply was, "She has had no return of the convulsions."

CASE II.—*Epilepsy.—Slight hypermetropia.—Ciliary Spasm.—Cure.*

MARY D., æt. 22; domestic servant. Sent by Dr. Morris. When 12 years of age was troubled with headaches and dimness of sight, and attended the Liverpool Eye and Ear Infirmary for a short time. Her first fit occurred in June 1885, and she had four on that day; for the next two or three months the attacks occurred daily, they then diminished in frequency to one weekly, and later on to one monthly until the beginning of January 1886.

Mar. 19, 1886.—*Ocular examination.*

Right eye . . . . [illegible] J 1 at 6 in.
Left „ . . . . [illegible] J 1 „

Mar. 26 (after atropisation for a week)—

Right eye . . . . Sph. + 1·25 D = [illegible]
Left „ . . . . Sph. + 1·25 D = [illegible]

Ordered + 0·50 D for near work. Atropine to be continued locally once daily for a fortnight.

Dec. 22, 1887.—No recurrence of fits. Can now read [illegible] with each eye without her glasses; she always uses these for working.

Sept. 1, 1888.—Remains well. Has had no more fits.

CASE III.—*Epilepsy.—Hypermetropia.—Relieved.—Return of fits with discontinuance of glasses.*

BERTHA B., æt. 16; nurse; came under notice Dec. 30, 1886. The history was that four months previously she had commenced to suffer from pain in the centre of her forehead accompanied with a feeling of numbness in the nose. On Nov. 21, 1886, at 9 P.M. she fell down in a fit, and seven of these occurred between that time and 1 A.M. Nov. 22. The fits recurred on Nov. 26, when she had four, and again on Dec. 22, when she had three more.

Dec. 30.—*Ocular examination.*

Right eye . . . $\frac{20}{20}$ (perfectly) sph. + 1·75 D = $\frac{20}{20}$ p.
Left „ . . . $\frac{20}{15}$ „ sph. + 1·50 D = $\frac{20}{15}$ p.

Jan. 6, 1887 (after atropisation for seven days)—

Right eye . . . $\frac{20}{0}$ sph. + 4·50 D = $\frac{20}{15}$
Left „ . . . $\frac{20}{0}$ sph. + 4·25 D = $\frac{20}{15}$

Jan. 27.—Ordered glasses, right + 3·50 D, left + 3·25 D, for constant wear.
Feb. 20.—Both eyes $\frac{20}{20}$ (with glasses).
Mar. 14.—Patient returned to service.

From the date of being fitted with glasses, she wore them regularly up to the middle of May (1887), and during that time she had not a single fit. She then discontinued the constant use of the glasses, and only wore them occasionally; the result was, that at the end of May the fits recurred, and from that time until the end of the year she had them frequently and severely. Last February she gave up the use of the glasses altogether, and the fits continued to recur, at any rate until the end of May.

In this case the patient certainly seemed relieved by the glasses, since for 3½ months after commencing to wear them she had no fits; whilst these returned with the discontinuance of the glasses. The case illustrates the difficulty of getting young women to wear glasses when their sight is not obviously defective to themselves.

CASE IV.—*Epilepsy of fourteen years' duration.—Hypermetropic astigmatism.—No improvement.*

MARY ELLEN C., æt. 29; married; one child; has had fits since 15 years of age.

Jan. 20, 1888.—*Ocular examination.*

Right eye . . . $\frac{20}{100}$ (1 letter) $\frac{20}{70}$ (1) J 6
Left „ . . . $\frac{20}{30}$ (4 letters) $\frac{20}{20}$ (4) J 1

Jan. 23.

Right eye . . . Cyl. + 2 D (axis 60°) = $\frac{20}{20}$ (perfectly)
Left „ . . . $\frac{\text{Sph.} + 0\cdot25\ \text{D}}{\text{Cyl.} + 1\cdot25\ \text{D (axis } 60^\circ)} = \frac{20}{20}$ „

Full correction ordered.

Aug. 23.—Patient has worn her glasses constantly since above date, but no improvement has resulted to the epilepsy, the fits continuing as frequently as before the case came under treatment. Since the epilepsy was of fourteen years' duration improvement was hardly to be expected.

CASE V.—*Epilepsy.—Compound hypermetropic astigmatism.—Cure.*

MISS CLARA P., æt. 21.

History.—At the age of 18 became a school teacher, but finding that evening study induced headache, she gave it up at the end of twelve months, and her headaches ceased simultaneously. After remaining at home for five months, during which time she was free from headache, she took the situation of a shop book-keeper, and her headaches recurred almost at once. On Aug. 23, 1887, whilst engaged in her ordinary work, she fell down unconscious in a fit, which her medical attendant (Dr. White of Warrington) states was an attack of epilepsy (*haut mal*). This was the first fit she ever had.

Sept. 17, 1887.—*Ocular examination.*

Right eye . . . $\frac{20}{70}$ (2 letters) $\frac{20}{50}$ (3) $\frac{20}{40}$ (2) J 1 (not well)
Left „ . . . $\frac{20}{30}$ (3 letters) $\frac{20}{20}$ (1) J 1 (fairly well)

Sept. 26 (after atropisation for a week)—

Right eye . . . $\frac{\text{Sph.} + 3\ \text{D}}{\text{Cyl.} + 1\cdot50\ \text{D (axis } 120^\circ)} = \frac{20}{20}$ (perfectly)
Left „ . . . $\frac{\text{Sph.} + 2\cdot25\ \text{D}}{\text{Cyl.} + 1\cdot25\ \text{D (axis } 120^\circ)} = \frac{20}{20}$ „

Ordered glasses for constant wear.

Right eye . . . $\frac{\text{Sph.} + 1\cdot50\ \text{D}}{\text{Cyl.} + 1\cdot50\ \text{D (axis } 120^\circ)}$

Left „ . . . $\frac{\text{Sph.} + 0\cdot75\ \text{D}}{\text{Cyl.} + 1\cdot50\ \text{D (axis } 120^\circ)}$

On May 18, 1888 (eight months after being fitted with glasses) patient wrote to say that she had had no recurrence of the fit, her headaches were so slight as to be scarcely worth mentioning, and that she had never felt so well in all her life.

Sept. 1, 1888.—Remains well. No recurrence of fit.

CASE VI.—*Epilepsy.—Compound hypermetropic astigmatism.—* *—Relieved* (?).

ISAAC P., æt. 14; sent by Dr. Bushby of Liverpool; has suffered from internal strabismus of left eye since the age of two. For the past 22 months has followed the occupation of an upholsterer. On Sept. 9, 1887, had a genuine epileptic fit; this was repeated on Nov. 20, and again on Nov. 30.

Nov. 29, 1887.—*Ocular examination.*

Right eye . . . . . . $\frac{20}{30}$ (2 letters)
Left „ . . . . . . $\frac{20}{0}$

Dec. 1 (after atropisation for two days)—

Right eye . . . $\frac{\text{Sph.} + 5\cdot50\ \text{D}}{\text{Cyl.} + 0\cdot50\ \text{D (axis vert.)}} = \frac{20}{30}$ (2 letters)

Left „ . . . $\frac{\text{Sph.} + 5\cdot50\ \text{D}}{\text{Cyl.} + 1\ \text{D (axis } 120^\circ)} = \frac{20}{0}$

Glasses ordered for constant wear.

Right eye . . . $\frac{\text{Sph.} + 4\ \text{D}}{\text{Cyl.} + 0\cdot50\ \text{D (axis vert.)}}$

Left „ . . . $\frac{\text{Sph.} + 4\ \text{D}}{\text{Cyl.} + 1\ \text{D (axis } 120^\circ)}$

Dec. 20 (with glasses)—

Right eye . . . . . . $\frac{20}{30}$ (perfectly)
Left „ . . . . . . $\frac{20}{200}$ J 16.

Dec. 21.—Resumed work.

1888. Had one fit on each of following days:—Jan. 22, April 5, May 12 and 19, June 17, July 7.

Aug. 2.—No fit since July 7.

Right eye . . . $\frac{20}{20}$ (perfectly)
Left " . . . . $\frac{20}{100}$ (1 letter) $\frac{20}{70}$ (1) J 8 (not well)

This case is still under observation, and it is yet too soon to pronounce a decided opinion upon it; but as the patient had three fits in seven weeks before being fitted with glasses, and these have since then diminished to six in eight months, there seems reason for thinking that some relief has been afforded.

CASE VII.—*Epilepsy.—Simple hypermetropic astigmatism.—No improvement.*

HENRY H., æt. 19; sent by Dr. Bushby.

On July 22, 1884, was three and a half hours in a swing-boat, and on the following day (July 23) had his first fit; had no more fits that year, but two occurred in the following year, two in 1886, two in 1887, and two in 1888 up to the time he was fitted with glasses.

Feb. 3, 1888.—*Ocular examination.*

Right eye . . . $\frac{20}{50}$ (5 letters) $\frac{20}{30}$ (1)
Left " . . . . $\frac{20}{50}$ (5 letters) $\frac{20}{30}$ (4)

Feb. 13 (after atropisation for a week)—

Right eye . . . Cyl. + 1·25 D (axis vert.) = $\frac{20}{10}$ (perfectly)
Left " . . . . Cyl. + 1·25 D (axis vert.) = $\frac{20}{10}$ "

Continued the use of atropine up to Feb. 19, when he began to wear his glasses. So far no improvement whatever has resulted therefrom; he has had numerous attacks of *petit mal*, alternating with decided fits, seizures of the former kind occurring pretty frequently, of the latter about once a month. Since the epilepsy in this case was of three and a half years' standing before the case came under notice, it could not be considered a favourable one for treatment.

CASE VIII.—*Epilepsy.—Myopia over-corrected with glasses.—Relieved.*

MR. ——, æt. 30: had a first epileptic fit when 18 years of age, and since that time he has had twelve such attacks, these occurring on an average about once a year; the last fit he had was on July 31, 1887, the day after visiting the Manchester Exhibition. He first wore glasses for myopia at the age of 16, and has since then twice increased the strength of the lenses. He came under notice on Aug. 19, 1887, and it was then found that he was wearing glasses two dioptres too strong.

Ocular examination—

Right eye . . . $\frac{20}{0}$ sph. $-3\cdot25$ D $= \frac{20}{30}$ (perfectly) $\frac{20}{20}$ (5)
„ $-3\cdot50$ D $= \frac{20}{20}$ „
Left „ . . . $\frac{20}{0}$ sph. $-4\cdot25$ D $= \frac{20}{30}$ „
„ $-4\cdot50$ D $= \frac{20}{20}$ „

Ordered, right, sph. $-3\cdot50$ D; left, sph. $-4\cdot50$ D. The glasses he had been wearing were, sph., $-5\cdot50$ D. each eye.

Aug. 26, 1888.—Since wearing the corrected glasses, patient has not had a single fit, that is an interval of upwards of a year has elapsed; so that we have ventured to put the case down as relieved; since, however, as long an interval has elapsed on former occasions, it is too soon to pronounce positively on the case. Patient has taken the bromides pretty regularly ever since his first attack, but less so of late than formerly.

CASE IX.—*Epilepsy.—Hypermetropic astigmatism.*

ELIZABETH R., æt. 19, came under the care of Dr. Davidson at the Liverpool Royal Infirmary, for epilepsy, to which she had been subject for two and a half years. There was a history of epilepsy in the family. The first fit, which occurred in November 1885, was immediately induced by a fall, in which her head was struck, and she had a series of fits, about forty in all, that day and the following; since that date they have recurred at pretty frequent intervals. The fits continued much as usual during the time she was under treatment at the Infirmary.

On Aug. 17, 1888, an ocular examination was made at Dr. Davidson's request.

Right eye . . . $\frac{20}{30}$ (4 letters) J 1 (fairly)
Left „ . . . $\frac{20}{30}$ (4 letters) J 1 (fairly)

Aug. 24 (after atropisation)—

Right eye . . . $\frac{\text{Sph.} + 0{\cdot}75\ \text{D}}{\text{Cyl.} + 1\ \text{D (axis vert.)}} = \frac{20}{20}$ (perfectly)

Left " . . . $\frac{\text{Sph.} + 0{\cdot}75\ \text{D}}{\text{Cyl.} + 1{\cdot}25\ \text{D (axis vert.)}} = \frac{20}{20}$ "

Aug. 31.—Commenced to wear glasses.

Since this patient has only just begun the use of the glasses, it is manifestly too soon to give any opinion as to the result.

Briefly now to sum up the results of the foregoing cases. In two of these (Nos. 4 and 7) no improvement whatever has resulted; in each of these cases the epilepsy was of some years' duration, viz. 14 and 3½ years, respectively. Number 9 is of too recent date to permit of any conclusions being drawn with reference to it, and hence, in calculating results, it must be struck out. One case (No. 3) discontinued treatment, and hence we think it safer to eliminate it altogether, since, though relief was afforded in the first instance, it is manifestly impossible to assert that had the patient continued to wear the glasses, she would have remained free from fits. Two cases (Nos. 6 and 8) we look upon as relieved. The remaining three (Nos. 1, 2, and 5) we have ventured to put down as cured, no fits having occurred during periods varying from ten months to two years and five months. To No. 2, indeed, we think this term may fairly be applied; whilst in Nos. 1 and 5 we must wait for a longer period to see whether the cure is permanent, since intervals of this duration between fits not unfrequently occur, especially at the first onset of the affection. Out of seven cases then in which the results are fairly definite, we have two patients unimproved, two relieved, and three cured, so far as present appearances go. These cases are of course far too few to permit of any definite conclusions being drawn; the results are suggestive, but in no way decisive. Taken, however, in connection with the large proportion of refractive errors in epileptic lunatics, which we think we have demonstrated, they seem to us to justify the assumption, that certain cases of epilepsy do occur in which the attack is induced by the undue strain put upon the muscular apparatus of the eye by reason of an abnormality of refraction. This being so, if the refractive error is corrected with glasses, there is reason to anticipate that the fits will subside, provided that this treatment is resorted to at a sufficiently early period of the case.

Future researches will doubtless determine the correctness or the reverse of this conclusion.

POSTSCRIPT.

Since the data for this paper were collected, and our notes put into shape, our attention has been called (by a review in the 'British Medical Journal,' May 5th, 1888) to a work recently published in America, by Dr. Stevens of New York,[1] which in part traverses the ground we have gone over. Into the general subject of functional nervous diseases we cannot of course now enter, but the section on epilepsy has much interest for us, inasmuch as we find that the author's researches confirm our own conclusions independently drawn. Dr. Stevens has indeed found a considerably larger proportion of refractive errors in epileptics than has come under our own cognisance, only eighteen out of one hundred consecutive cases being emmetropic. Several interesting cases are detailed, in which the correction of the refractive error by appropriate glasses was followed by the complete cessation of the epileptic fits, whilst in other cases in which the cure was not absolute, considerable amendment ensued. In some of the cases, however, tenotomy of one of the recti muscles, owing to insufficiency of its opponent, was practised in addition.

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[1] 'Functional Nervous Diseases; their Causes and their Treatment,' by Geo. T. Stevens, M.D.

TABLE SHOWING THE KIND AND AMOUNT OF REFRACTIVE ERROR IN FORTY-EIGHT CASES OF EPILEPTIC INSANITY, IN WHICH THE REFRACTION WAS ABNORMAL.

No.	Initials.	Sex.	Age.	REFRACTION.
1	J. H.	M.	31	High hypermetropic astigmatism
2	J. S.	M.	31	Compound hypermetropic astigmatism— Right eye . . $\frac{\text{Sph.} + 2\cdot25\text{ D}}{\text{Cyl.} + 0\cdot50\text{ D (axis vert.)}}$ Left „ . . $\frac{\text{Sph.} + 2\cdot25\text{ D}}{\text{Cyl.} + 0\cdot50\text{ D (axis vert.)}}$
3	E. C.	M.	25	Right.—Slightly myopic Left.—Slight mixed astigmatism
4	T. C.	M.	34	Simple hypermetropic astigmatism
5	J. H.	M.	29	Compound myopic astigmatism— Right . . . $\frac{\text{Sph.} - 10\text{ D}}{\text{Cyl.} - 3\text{ (axis }60^\circ)}$ Left. . . . $\frac{\text{Sph.} - 12\text{ D}}{\text{Cyl.} - 3\text{ D (axis }45^\circ)}$
6	T. D.	M.	30	Myopia.—Right – 2 D. Left – 2 D
7	T. D.	M.	51	Simple hypermetropic astigmatism— Right . . . Cyl. + 2·50 D Left. . . . Cyl. + 2 D
8	P. C.	M.	42	Hypermetropia.—Each eye + 2 D
9	R. A.	M.	39	Simple myopic astigmatism
10	J. B.	M.	42	Right.—Slight hypermetropic astigmatism Left.—Emmetropic
11	R. B.	M.	21	Right.—Myopic astigmatism Left.—Myopia
12	J. B.	M.	52	Hypermetropia.—Each eye + 2 D
13	E. D.	F.	34	Compound hypermetropic astigmatism— Right . . $\frac{\text{Sph.} + 3\text{ D}}{\text{Cyl.} + 3\cdot50\text{ D (axis vert.)}}$ Left. . . $\frac{\text{Sph.} + 3\text{ D}}{\text{Cyl.} + 3\text{ D (axis vert.)}}$
14	S. H.	F.	28	Myopia.—Right – 3·50 D. Left – 2·50 D
15	F. A.	F.	17	Hypermetropic astigmatism— Right . . $\frac{\text{Sph.} + 0\cdot25\text{ D}}{\text{Cyl.} + 1\cdot25\text{ D (axis vert.)}}$ Left. . . $\frac{\text{Sph.} + 2\text{ D}}{\text{Cyl.} + 1\text{ D (axis vert.)}}$
16	J. T.	M.	33	Simple hypermetropic astigmatism
17	G. S.	M.	31	Hypermetropia.—Right + 1 D. Left + 2 D
18	W. L.	M.	63	Hypermetropia.—Right + 2·50 D. Left + 2·50 D
19	H. K.	M.	17	Hypermetropia.—Each eye + 1·50 D
20	T. F.	M.	32	Hypermetropic astigmatism— Right . . $\frac{\text{Sph.} + 0\cdot50\text{ D}}{\text{Cyl.} + 4\cdot50\text{ D (vert.)}}$ Left. . . Cyl. + 1·50 D (vert.)
21	T. T.	M.	35	Hypermetropia.—Each eye + 1·25 D

No.	Initials.	Sex.	Age.	REFRACTION.
22	H. P.	M.	35	Compound hypermetropic astigmatism— Right . . . $\frac{\text{Sph.} + 1\cdot75\text{ D}}{\text{Cyl.} + 0\cdot75\text{ D (axis horizontal)}}$ Left . . . $\frac{\text{Sph.} + 1\cdot75\text{ D}}{\text{Cyl.} + 0\cdot75\text{ D (axis } 30^\circ)}$
23	E. P.	M.	44	Right.—Simple myopic astigmatism— Cyl. − 2·50 D (vert.) Left.—Compound myopic astigmatism— $\frac{\text{Sph.} - 3\text{ D}}{\text{Cyl.} - 5\text{ D (axis vert.)}}$
24	E. C.	M.	39	Hypermetropia.—Right + 2·25 D. Left + 2 D
25	E. G.	M.	29	Compound hypermetropic astigmatism— Each eye . . $\frac{\text{Sph.} + 3\text{ D}}{\text{Cyl.} + 1\cdot50\text{ D (axis vert.)}}$
26	J. N.	M.	38	Hypermetropia.—Each eye + 1·25 D
27	J. J.	M.	17	Hypermetropia.—Each eye + 2 D
28	C. M.	F.	39	Right.—Simple myopic astigmatism. Left.—Emmetropic
29	M. S.	F.	30	Hypermetropic astigmatism— Right . . $\frac{\text{Sph.} + 0\cdot50\text{ D}}{\text{Cyl.} + 1\text{ D (axis horizontal)}}$ Left . . $\frac{\text{Sph.} + 0\cdot75\text{ D}}{\text{Cyl.} + 0\cdot75\text{ D (axis horizontal)}}$
30	J. P.	F.	37	Right.—Hypermetropic astigmatism— $\frac{\text{Sph.} + 1\text{ D}}{\text{Cyl.} + 0\cdot75\text{ D (axis } 30^\circ)}$ Left.—Emmetropic
31	A. C.	F.	25	Compound hypermetropic astigmatism— Each eye . . $\frac{\text{Sph.} + 2\cdot50\text{ D}}{\text{Cyl.} + 1\cdot50\text{ D (axis vert.)}}$
32	A. M.	F.	9	Hypermetropia.—Each eye + 8 D
33	E. C.	F.	25	Hypermetropic astigmatism— Right . . $\frac{\text{Sph.} + 1\text{ D}}{\text{Cyl.} + 0\cdot75\text{ D (axis } 120^\circ)}$ Left . . . $\frac{\text{Sph.} + 1\text{ D}}{\text{Cyl.} + 0\cdot75\text{ D (axis vert.)}}$
34	F. K.	M.	35	Hypermetropia.—Each eye + 2 D
35	W. T.	M.	27	Hypermetropic astigmatism— Right . . $\frac{\text{Sph.} + 1\text{ D}}{\text{Cyl.} + 0\cdot75\text{ D (axis horizontal)}}$ Left . . . $\frac{\text{Sph.} + 1\text{ D}}{\text{Cyl.} + 0\cdot50\text{ D (axis horizontal)}}$
36	P. B.	M.	19	Hypermetropia.—Each eye + 9 D
37	E. H.	M.	66	Simple hypermetropic astigmatism
38	A. S.	F.	25	Hypermetropia.—Each eye + 2 D
39	C. H.	F.	6	High hypermetropia
40	C. W.	F.	45	Simple hypermetropic astigmatism
41	S. W.	F.	46	Slight myopia

No.	Initials.	Sex.	Age.	Refraction.
42	E. W.	F.	50	Hypermetropia.—Right + 3·50 D. Left + 4·50 D
43	A. M.	F.	29	Hypermetropic astigmatism— Each eye . . $\frac{\text{Sph.} + 1\text{ D}}{\text{Cyl.} + 1\cdot75\text{ D (axis vert.)}}$
44	E. W.	F.	23	Hypermetropia.—Each eye + 1·75 D
45	C. W.	F.	38	Compound myopic astigmatism— Right . . $\frac{\text{Sph.} - 2\text{ D}}{\text{Cyl.} - 1\cdot50\text{ D (axis } 120^\circ)}$ Left . . . $\frac{\text{Sph.} - 3\text{ D}}{\text{Cyl.} - 1\cdot50\text{ D (axis horizontal)}}$
46	E. C.	F.	42	Right.—Simple hypermetropic astigmatism— Cyl. + 1·50 D (axis 120°) Left.—Simple myopic astigmatism— Cyl. − 1 D (axis horizontal)
47	N. B.	F.	28	Hypermetropia.—Right + 1·75 D. Left + 1·50 D
48	A. T.	M.	38	Simple myopic astigmatism— Right . . . Cyl. − 4 D (axis vert.) Left . . . Cyl. − 2·50 D (axis vert.)

XEROSTOMIA (DRY MOUTH).

BY W. B. HADDEN, M.D. LOND., F.R.C.P.,

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ONLY a few examples of this affection have as yet been described. The subject was brought before the Neurological Society, and a patient exhibited in 1887 by Mr. Hutchinson. Next year an example came under my own notice, and the history of the case, together with Mr. Hutchinson's, are recorded in the Clinical Society's Transactions, vol. xxi.

The term "Xerostomia" (ξηρός, dry, στόμα, mouth) has recently been suggested for this condition by Mr. Hutchinson and myself independently.

A brief account of the disease, so far as its causation and symptoms are as yet known, may prove of interest. All the subjects were women, and most of them were between the ages of fifty and sixty-five; but in one the onset occurred at the age of twenty-three, in another at thirty-five, and in another at forty-four. The disease began suddenly in three cases, preceded in two by severe mental shock.

In all the cases the condition of the mouth is the same. The tongue is red, devoid of epithelium, cracked, and absolutely dry; its appearance is like that of raw beef. The inside of the cheeks, the hard and soft palate are also dry, and the mucous membrane smooth, shiny, and pale. The salivary glands appear normal, and no mechanical obstruction has been detected in their ducts. The general health remains unimpaired, and in spite of the absence of saliva, no digestive disturbance is noted by any of the observers. Articulation, however, is difficult, in consequence of the absence of moisture, and swallowing has to be assisted by constant sipping. Such are the symptoms which are common to all the cases. It must also be added that the disease was neither preceded nor accompanied by any special nervous symptoms, with the exception of the nervous shock at the initial period in the two cases already alluded to.

There are some additional points which may or may not prove to be important subsequently. Some of these symptoms have simply been recorded in isolated cases, or have received attention from two or three observers only.

In two cases there was dryness or deficiency of moisture of the pharynx; in two the secretion from the nose was diminished; in two the lachrymal secretion was arrested; and in two the skin was dry. In two instances falling out of the teeth is recorded.

Taste seems to be natural when substances are in solution. In my own case common sensation of the inside of the mouth and on the tongue was unimpaired, and the reflex action of the palate was present.

The urine when examined has been found normal, and it is expressly stated in two cases that sugar was not present.

Of this affection Mr. Hutchinson appropriately remarks: "The condition appeared to me to be an exaggeration assuming permanency, of the state of mouth which is common under the sensation of fear, and during states of nervous excitement." This practically embodies the view which I briefly put forward in the Clin. Soc. Trans. vol. xxi., viz. that the suppression is not dependent on gross change in the glands, but should probably be ascribed to some disorder of the nervous apparatus. When my patient first came under observation, I made enquiries with the object of ascertaining if there was known to physiologists any one centre controlling the secretion of all the salivary and buccal glands. Though experimental observation is silent on this point, there can be no doubt that such a centre does exist. This view is put forward by Mr. Hutchinson in the 'Illustrated Medical News,' Jan. 26th, 1889, p. 76.

The disease reaches its greatest intensity rapidly, and sometimes suddenly, and then remains without change for years. Such seems to be the rule. In my own case the administration of jaborandi, in doses of thirty to forty minims three times daily, had a very beneficial effect, the mouth becoming moist about a quarter of an hour after each dose. I omitted the drug for a time, but the patient soon returned, and asked me to give it her again. Dr. L. Morgan[1] has also noted great improvement under jaborandi. Pilocarpin, in my experience, is less satisfactory; and this again is in accordance with Dr. Morgan's experience. Mr. Butlin[2] found benefit from hydrochlorate of pilocarpin in doses of $\frac{1}{12}$ of a grain. It is right to mention that according to Mr. Hutchinson and others, jaborandi was administered without any success. Mercury, quinine, steel and opium have been tried ineffectually. Dr. Broadbent has found iodide of potassium of some use. The application of glycerine to the mouth is of decided value. To my own patient I suggested a plentiful use of horseradish; but I could not satisfy myself that it was of service.

NOTE.—Since writing the above, I have come across an allusion to this condition of Dry Mouth in Dr. Weir Mitchell's 'Lectures on Diseases of the Nervous System' (Second Edition, p. 209). The patient, a woman of thirty-one, suffered from aggravated hysteria, such as retention of urine, stupor, paralysis, hyperæsthesia and aphonia. At one time she had anuria, and then "complete absence of saliva, so that for a time the mouth was absolutely dry."

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[1] 'Medical Press and Circular,' January 2, 1889, p. 4.

[2] See Report of Clinical Society, 'Brit. Med. Journ.' Nov. 3, 1888.

THE EPILEPTIC PAROXYSM.

SUPPLEMENTARY PARAGRAPH.[1]

BY JAMES OLIVER, M.D., F.R.S. (EDIN.).

Tongue-biting.—Evidence of tongue-biting, although of some worth as an aid in the diagnosis of epilepsy, is not, to my mind, an infallible manifestation—when present—of the disorder in question. On several occasions I have witnessed attacks in women in which the movements were typically hysteroid or purposive in character, and yet these patients, after many of the convulsive paroxysms, revealed a laceration of the tongue. It might, of course, be suggested, and plausibly too, that the tongue-biting in such cases was a mere casual coincidence. The fact, however, that its recurrence was frequent dispels, to a certain extent, the feasibility of mere accidental association. If there be any slight dissociation in the movements of the lower jaw and tongue in death, as occasionally happens in the act of mastication, the tongue may be, and frequently is, bitten. In epileptic and other convulsive attacks, tongue-biting is probably the result of lack of harmony in the movements of the tongue and lower jaw.

Evidence of laceration of the tongue is not so commonly noted as many authors would lead us to believe, and probably after all, when considered with the frequency of attacks, is a mere coincidence.

As far as my own observations go I am convinced that the tongue is always bitten on that side which is the seat of initial motor spasm; this side corresponds with that towards which the head and eyes are primarily directed. It is to be remembered, however, that the head is sometimes during the attack deviated—rapidly it may be—from side to side, or directed primarily to one side, and it may even, throughout the greater part of the actual attack, be deviated to the side away from that which evinced initially the motor spasm. In noting, therefore, the relationship of tongue-biting with deviation of the head and eyes, the attack must be witnessed from the beginning, as the head may, at any period of an epileptic attack, change its direction. Foot clonus, as I have elsewhere remarked, can often be elicited after an epileptic paroxysm, and I have invariably noted that this phenomenon is more marked, and more readily produced on the side corresponding with that on which the tongue is lacerated. Now foot clonus is more easily developed, and continues longer in existence on that side on which the convulsion was greater, and this is usually that first attacked.

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[1] [To be read in connection with the author's paper (see page 343 of the present volume), from which it was accidentally omitted.—ED.]

Regarding the mechanism of tongue-biting in epilepsy, many suppositions have been entertained as explanatory. It has been alleged that the tongue, during the actual fit, becomes engorged because of an interference with the return of blood, and that the consequent increase in size of the organ is the cause of occasional laceration, the tongue thereby projecting between the teeth if the lower jaw should happen at any time during the attack to be depressed. If, however, tongue-biting during an epileptic seizure were due to mere engorgement and swelling of the organ, one would expect to find it bitten more frequently on both sides during the same attack. Lately I saw a patient in whom the muscles of the right half of the tongue had gradually atrophied, and during the early days of this slowly progressive change the tongue was frequently bitten on the right side during mastication. In this case the muscles of the left half of the tongue being inefficiently opposed by those of the right, thrust the organ between the teeth, and thereby laceration of the right half resulted. Adaptation to the structural change eventually ensued, and now, although the atrophy of the right half is most marked, the tongue seldom is bitten. Some authorities state that laceration of the tongue during the epileptic fit is due to the organ being protruded beyond the teeth, and thereby caught.

Protrusion of the tongue is a movement habitually associated with depression of the lower jaw, and consequent elevation of the hyoid bone. If, therefore, the protrusive theory be accepted, the organ can only be lacerated when the movements are dissociated. The fact, however, that the tongue is invariably bitten on that side on which the motor spasm is first manifested tends to thwart this view. If laceration occurred during the act of protrusion the organ would necessarily be bitten on the side away from that which was the seat of motor spasm. That the tongue is bitten by a process of retraction and dissociation of movements I have no misgivings.

Clinical Cases.

A CASE OF ATAXIC PARAPLEGIA, WITH AUTOPSY.

BY CHARLES L. DANA, A.M., M.D. (NEW YORK).

Most cases of ataxic paraplegia are cases of locomotor ataxia with extension of the process; or cases of lateral sclerosis with extension of the process. These latter cases originate, as a rule, in a somewhat diffuse transverse myelitis affecting chiefly the two lateral columns, and from this point secondary degenerations extend.

There are sometimes cases of primary sclerosis of all the long-fibre columns of the cord.

Such are about the views expressed by me in a paper on "Spastic Ataxia"[1] read before the New York Academy of Medicine two years ago ('New York Medical Record,' July 2, 1887), and in a discussion in Dr. Putnam's paper on this subject a year ago.

The following case illustrates the pathology of one form of this affection very well.

The following is the outline of the history:

Male, age 50.—Syphilitic infection in 1863, acute and chronic lead poisoning, colic, left facial paralysis, amblyopia, iritis. Gradual onset of a spastic paraparesis, with later development of ataxia, until spastic, and ataxic, and paretic symptoms were about equally prominent. Sphincters not involved. Some anæsthesia, little pain.

Death from diarrhœa.

Autopsy.—Lateral sclerosis of entire length of cord, most marked in mid-cervical region; slight posterior sclerosis in cervical region; chronic lepto-meningitis.

Nerve roots and peripheral nerves normal.

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[1] In my original paper on this disease ('Medical Record,' July 2, 1887) I used the name "spastic ataxia," because in all my cases, which number now over fifteen, I have found the chief symptoms to be spastic ataxia, while the paralysis was not marked, there being paraparesis only. Dr. Gowers, however, first published the description of the symptoms, and is entitled to christen his child, though my own article was written before his was published.

I adopt the name "ataxic paraplegia," therefore, in the present case.

Jos. M., U.S., 50, single, painter, was admitted to my service in Bellevue Hospital, May 1st, 1888. He gave the following history: His occupation was that of painter.

In 1863 he contracted syphilis, and had secondary symptoms.

In 1870 he had a sudden temporary left facial paralysis.

In 1875 he had an attack of rheumatism (or lead poisoning).

In 1882 he had an attack of lead colic lasting six weeks.

In 1883 he suddenly lost his sight while at work. He improved very much in two months.

In 1884 he had an attack of colic lasting one day.

In 1885 he was admitted to hospital suffering from lead colic. He also had paræsthesia of right side of face and right leg, and headache.

Physical examination at this time showed a feeble paraparetic gait, but no ataxia in arms or legs, no anæsthesia or analgesia. Slight blue line on gums. The deep reflexes were decidedly exaggerated, the superficial normal.

Electrical examination of the muscles of the thighs and legs showed that they reacted with abnormal readiness to the faradic current (with secondary coil, slide at 0). With the galvanic current the muscles reacted with from 12 to 16 Leclanché elements, the anode requiring from 2 to 6 more elements than kathode. (There was at the time no milleamperemeter in the hospital.) Nothing especial could be learned from the examination.

Patient was re-admitted to hospital October, 1886, and an operation for irido-choroideotomy performed.

Patient entered the hospital again in May, 1888, and then first came under my care.

His past history, as seen, was that of chronic lead poisoning, and nervous syphilis, and irido-choroiditis.

Physical examination at this time showed no marked lesions outside the nervous system and eyes.

Intelligence good. His gait was feeble and uncertain; but he could walk alone with care. His legs were weak, and decidedly paretic. There was marked ataxia, static and locomotor. The deep reflexes were greatly exaggerated, ankle clonus present, and cramps in the legs occurred.

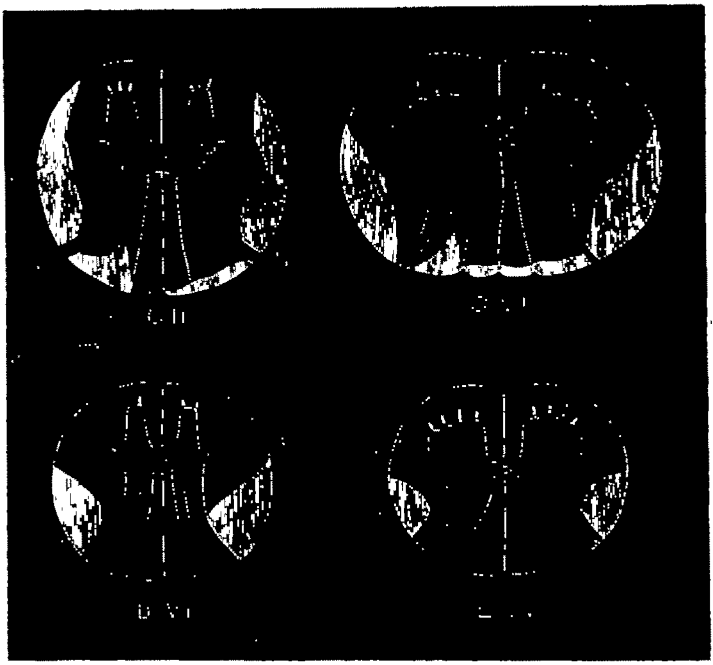
There was no special atrophy or paralysis of any muscle group.

There was a moderate degree of anæsthesia, tactile and pathic, in the legs below the knees, but not in the arms. The arms, however, showed ataxia even more than the legs.

Later in his disease he had a constant and severe pain in the right shoulder and down the back.

The bladder and rectal sphincters were normal. He had some pains in the back, and occasionally cramps and pains in the legs and shoulders.

He was shown to my class at the Post-graduate School as a very good example of ataxic paraplegia or combined sclerosis, the salient symptoms being—Paraparesis and arm weakness, a spastic condition of limbs, with exaggerated reflexes and ankle clonus; later, ataxia and anæsthesia, with occasionally some pains in shoulders, and arm and back. No sphincter troubles.



Diagrams showing sclerotic areas in the cord. Posterior leptomeningitic alterations not shown.

The patient continued about the same until midsummer of 1888, when he died of an intercurrent diarrhœa.

Autopsy.—Body considerably emaciated. Thoracic organs not examined. Kidneys small and granular.

Brain.—Dura mater thickened in places, and adherent to brain over region of pericranian bodies. Cerebral arteries thickened and patent. No meningitis or other abnormalities.

Spinal cord.—Dura mater thickened, and some external adhesive pachymeningitis is present in lower dorsal region of cord posteriorly.

On section evidence of lateral sclerosis and thickened pia is apparent.

Microscopical examination.—There is a chronic lepto-meningitis affecting the whole cord. It is not of a high grade, but is rather a slowly formed thickening.

Sections of the cord were made at various levels and stained in Weigert's hæmatoxylon, in aniline blue, congo red, and ordinary hæmatoxylon.

There was found a well-marked lateral sclerosis throughout the cord from the first cervical to the sacral segments.

The process was most marked in the mid-cervical region, where upon one side is evidence of nerve-tissue destruction and a sclerotic patch.

In the cervical region the posterior columns are affected in parts somewhat deeply, and throughout with a marginal sclerosis.

Below the cervical region the lesion is chiefly and essentially a descending lateral degeneration, the anterior columns not being at all affected.

Numerous examinations were made of the anterior and posterior cervical roots and of the anterior tibial nerve, but with negative results. The anterior tibial nerve simply shows some increase in its connective tissue framework, but no parenchymatous change.

The following is a more minute record of the microscopical examination:

1st Cervical segment.—Membrane decidedly thickened, and, where torn off, a very thin layer of medullary tissue comes with it. It is a little thicker posteriorly, and especially near the exit of posterior root.

Blood-vessel walls thickened.

Posterior roots show a little connective tissue increase, but no notable loss of fibres.

White matter.—Moderate degree of degeneration of lateral and one posterior columns as indicated in cut. Increase of interstitial substance, but no actual sclerotic focus. Vessels walls thick.

Gray matter.—Great increase of connective tissue cells around central canal. A good many enlarged vessels with thick walls.

Cells of anterior horn are apparently diminished in number and size.

Mid-cervical segment.—Meningitis as above, but on each side more deeply adherent to lateral columns, and at one side a deep excavation is present where these membranes have been torn away. Anterior roots on each side here show no degeneration.

White columns.—On one side is an excavation produced by tearing away meninges, and here is found a marked sclerotic focus with degeneration of nerve fibres. A less marked degenerative area is seen on the other side.

The anterior columns are normal, including the antero-median.

The periphery of the posterior columns is decidedly affected, there being loss of nerve fibres without, however, any marked increase of connective tissue. It looks as if the change were due to imperfect nutrition from the inflamed meninges.

The grey matter.—The cells of the anterior horns are somewhat diminished in number. There is increase of connective tissue nuclei around the central canal and thickened blood-vessels.

The sclerotic and degenerative changes are decidedly more marked in the mid-cervical than at the first and second cervical segments, and the anterior columns were not at all affected. There can be no doubt, therefore, that the morbid changes were not secondary to cerebral lesions, although unfortunately the brain and medulla were spoiled and could not be examined.

In the dorsal and lumbar regions there is seen only the lepto-meningitis and lateral sclerosis. The gray matter and its cells are normal, including Clark's columns.

Sections of the anterior tibial nerve and of the anterior and posterior nerve roots showed no marked changes.

The pathological diagnosis is, therefore, lateral sclerosis, posterior marginal sclerosis in cervical region, chronic lepto-meningitis.

The clinical diagnosis, ataxic paraplegia.

CASE OF EPILEPSY, IN WHICH THERE ARE PERIODS OF AUTOMATISM OF A VERY WELL MARKED NATURE.

BY DR. GEO. H. SAVAGE, BETHLEM ROYAL HOSPITAL.

I SAW this patient first in 1886, and he had been occasionally under Dr. Hughlings Jackson for many years before this date. He was, when I first saw him, aged sixty-one, and married.

He had been a silversmith, and had recently retired from business.

There was no history of neurosis in his family, he was sober, had never been intemperate, nor had he had syphilis, there were no signs of injury to his head; he said he had had fits for thirty-four years. The history he gives of the first fit is as follows: that after a period of intense work and worry, he saw a hideous face start up before him, this vanished when he rested from his work, but on resuming it, the vision reappeared. After this followed a vision of a landscape, then he lost his senses for a time.

Ever since he has had fits; these occur chiefly by day and are without any cry, aura, or any hallucination, he sleeps as a rule after their occurrence, he has bitten his tongue once or twice severely, the fits are irregular in their intervals, but occur about once monthly. His speech at times has been affected after a fit, so that he was accused of drunkenness; he has no local pains, no loss of sight, or any special loss of mental power or of memory. The first mental disorder was change of feeling towards his wife; thus from being a very loving husband he began to suspect that she wanted to poison him; next he took a dislike to Dr. Jackson, saying that he was in league with his wife to poison him with digitalis. I formed the opinion that he was quite unfit to transact any business affairs at the time I saw him, and I gave him "bromidia," as he was sleepless. For two years his wife and daughter managed him at home, but his wife dying, he became hard to control, and during periods of unconsciousness, he signed cheques and gave away money without having any recollection of what he had done.

He was admitted into Bethlem Hospital on June 6, 1888, on medical certificates, stating that he believed there was a conspiracy among the doctors and others to murder him; he said portions of his body were being burnt in half moons.

The true epileptic fits were now rare, but memory defective, and he was full of suspicion. He is said to have more attacks of petit mal, and simple losses of consciousness—several of these may occur in a day; he had shown a tendency to wander from home without reason or recollection.

The circulation and respiration were normal.

Pupils equal and re-acting, fundus oculi healthy, tongue steady, slightly furred, appetite good, bowels regular. Knee reflex normal, speech slightly blurred. He said he felt perfectly well, he was restless and unable to keep long at one thing. One fit occurring a week after admission was thus reported: he did not cry out, both sides appeared equally affected, he did not bite his tongue or pass fæces or water involuntarily; after a short sleep he became restless, wanting to get out of bed, he was confused in manner, and his speech was unintelligible, pupils dilated, with no reaction to light, no unilateral weakness, skin perspiring freely.

Up to June 22 he had daily attacks of the petit mal; on this date pot. bromidi gr. xv. ter die was given, and this controlled the fits, none being noticed till July 29, when I was present during the fit, and I submit the following details:—

As I was walking through the ward, Mr. A. beckoned to me through a glass door, and when I joined him he appeared to be slightly excited and flushed. He asked me if I would sit down and listen to him; I agreed, and he began to talk about his former illnesses and the very great kindness shown by Dr. Hughlings Jackson; he was slightly emotional as he went on to speak of his visits to the doctor, then, without any warning, he passed on to talk quite irrelevantly about his late wife and of his sexual relations. At this time he pointed towards his penis with his right index finger. I noticed that he then looked fixedly before him as if in abstract thought. At first I fancied this was done to suppress his emotion at the thought of his late wife.

Then suddenly his head was turned to the right, and he began vigorously picking his left thumb-nail with his right thumb and first finger; after he had done this for a minute he turned his head over to his left shoulder—away from me—and began to pick his right thumb-nail so vigorously that I had to interpose to prevent its evulsion, yet evidently he did not feel any pain.

He desisted from his picking, got up without any hesitation or stagger, and walked slowly about the room; he walked to a window, which he opened and looked out of, then he turned round and watched the birds in an aviary, with apparent interest; every act had the appearance of design and care, he avoided obstacles and directed his steps easily and freely.

He walked to the door where he had first seen me and appeared to see some one, yet when I placed myself in his way he did not recognise me, but changed his direction to pass by me.

I touched him gently, but he took no notice, and when I spoke quietly also he did not react, but when I clapped him on the shoulder and spoke loudly at the same time, he answered as one waking from sleep. He then relapsed into his somnambulistic state and remained in it for ten minutes longer. In all he was nearly half an hour unconscious.

On my speaking to him after his return to consciousness he apologised for breaking off the conversation about Dr. Hughlings Jackson, and said he did not quite understand those lapses of consciousness, but that he thanked God he had now no fits. Altogether he has had some six or eight of similar though less well remarked attacks while in this hospital, and since July he has had one genuine epileptic fit.

In this case then we have a man who is subject to true epilepsy, and who has also very well marked periods of automatism, and who in some of these has squandered money, and in others for half an hour at a time he has performed highly complicated acts unconsciously.

The peculiar way in which the picking of first one thumbnail and then the other, associated with the rotation of the head, confirms Dr. Jackson's belief that these automatic periods are generally, if not always, post-epileptic.

The medico-legal importance of such states cannot be overestimated.

LOCOMOTOR ATAXIA OF AT LEAST FIVE YEARS' DURATION, WITH LARYNGEAL CRISES, AND CHARCOT'S JOINT-LESION, TERMINATING IN GENERAL PARALYSIS OF THE INSANE.

BY HENRY HANDFORD, M.D., M.R.C.P.,

Physician to the General Hospital, Nottingham.

LARGELY through the observations of Dr. Savage the association of locomotor ataxia with general paralysis of the insane has been established. It has been recently shown[1] that not only the cord, but also the sciatic and the peripheral nerves undergo degenerative changes in the vast majority of cases of general paralysis. A few cases evidently *commence* by a degeneration of the cord, or of the peripheral nerves, or of both; and some authorities consider that most do so. The majority sooner or later develop ataxic symptoms more or less; and in some the ataxia is well marked, while the mental alteration remains somewhat in abeyance. The occurrence of well-marked locomotor ataxia in the middle, or towards the latter part of a case of general paralysis, cannot be said to be altogether infrequent. But it is far from common for general paralysis to supervene upon a typical case of locomotor ataxia of several years' standing. The following is an example of this condition. Unfortunately no post-mortem examination was made, as the friends refused permission, notwithstanding the most strenuous endeavours to obtain their consent.

CASE I.—S. V., aged 28, married, a framework knitter, was admitted into the Nottingham General Hospital under my care in February 1886, complaining of difficulty of walking. His father was an insane epileptic, and died in an asylum. The patient used to bathe a good deal in the Trent, even in winter. No history of syphilis could be obtained. About 3½ years ago he began to experience difficulty in walking, and frequently used to fall down. His sight failed, and he had attacks of vomiting, and occasional loss of control over bladder and rectum. In 1883 he was an in-patient in this hospital, and was diagnosed to be suffering from locomotor ataxia. He then had retention of urine and required the use of a catheter. A few days before his admission on the second occasion, he fell and twisted his right knee, but felt no pain. "He often falls and bruises himself very much, but never feels any pain, and only finds out the bruises from seeing the discoloration of the skin." When I saw him, the knee presented the typical appearance of Charcot's joint lesion, and it remained very much in the same condition till his death 1½ years later. There could be no reasonable doubt that the joint lesion was started by injury, though, as will be seen later on, there was abundant clinical evidence of peripheral neuritis. On attempting to walk, the movements of the legs were most disorderly. After much oscillation he could at length stand fairly steadily with the eyes open, but fell at once when they were closed. In the left leg the knee-jerk was quite absent: on the right it could not be tested on account of the swelling of the knee. There was no ankle-clonus. On examining the muscular sense, it was found that he could not place the legs in any desired position, either with the eyes open or closed; and with the eyes closed he could not tell in what position the legs had been placed for him. But on attaching weights to the legs, he could readily detect a difference of 1lb., and in the arms, differences of a few ounces were easily perceived. He could distinguish correctly between a test tube filled with hot water and one filled with cold, both when applied to the legs and the arms. He could not feel at all a light touch with the finger on the legs. The prick of a pin could sometimes be felt like a touch, but without any pain. In the arms no loss of sensation was detected, though he complained that feeling was impaired. About six months before his admission the second time, the arms began to be affected. His hands and fingers, he said, "went dead," and he lost power in them. He frequently dropped things, and in lifting a glass or a cup, always had to use both hands. The lips, tongue, and pharynx were affected. He spoke very thickly, the lower lip dropped somewhat, and the mouth was slightly drawn to the left. There was loss of sensation in the mouth, so that he could not feel whether any fluid he wished to drink was in his mouth or not, neither could he tell whether the cup was touching his lips. After a short time, by rubbing the lips, &c., the feeling returned sufficiently to enable him to drink. At the time of his admission there was no muscular atrophy. During his stay in the hospital the gastric crises were marked, but infrequent. There were occasional shooting-pains but no incontinence of urine or fæces. While I was examining his throat, on his opening his mouth widely and throwing his head back to enable me to see better, he had a curious epileptoid attack, affecting the muscles of the face, lips, pharynx and larynx. The breathing was interfered with, the face became turgid and dusky, and the whole appearance was alarming. This condition lasted about half a minute. He said similar attacks used to come on when he held his head back to be shaved, much to the alarm of the barber. Sometimes the attacks last several minutes, and he falls, if standing at the time. He loses consciousness, and remembers nothing of what happens while he is in the attacks. When these epileptoid attacks could be avoided the vocal cords were seen very readily. During quiet respiration the left cord was further from the middle line than the right, and its border was concave, showing some paralysis of the inner fibres of the thyro-arytænoid muscle. Both cords moved during phonation, but the left less freely than the right. He had some difficulty in swallowing solid food, and had to drink with each mouthful to enable him to swallow it. The tongue was protruded to the left and the face was drawn to the left during articulation and during the epileptoid attacks. Taste was tested with quinine, salt and sugar. It was very deficient, but not absent. Common sensation in the tongue was much diminished. The power of smell was sluggish. There was partial ptosis of the left eye. Sight was imperfect. The pupils were unequal: the right was dilated to the size of a No. 10 catheter, and the left was about the size of a No. 6. They did not react to the strong light of a mirror, and it was doubtful if they moved at all with accommodation. The discs were grey, and the connective tissue stroma was too plainly visible. He slept badly and was low-spirited. He said that for ten months past he had occasionally, especially after reading or thinking, suddenly lost his memory and power of attention. "He wants to do or say something, but cannot tell what it is." If he had been reading, he did not know what he had been reading about, and could not understand what he continued to read, and had to stop. In about a quarter of an hour he was all right again. After a few weeks he left the hospital and went to live with his mother and a married sister, but he was so quarrelsome that the sister left, and he remained alone with his mother. He had some kind of fit or congestive attack, and shortly after this, began to grow excited and sleepless, and used to sit up all night. Though in receipt of parish relief he took a new house, began to drive about in cabs, and buy all sorts of things he could not pay for. He told his relations he was going to take them to a big house in the country, where he was going to build a lot of factories. He drove up to my house in a cab and said his mother had a lot of money, and had started a line of omnibuses. He himself had plenty of money, and he wanted to be a private patient. He was very anxious to have all his teeth out, as he thought he could eat better with an artificial set. He persisted in this desire for several months. His teeth were remarkably good and sound, and never showed that tendency to fall out, which is sometimes observed in cases of locomotor ataxia. Next day he drove up to the hospital and told them I had advised him to have his leg off, and to have a cork one instead, and he wanted that done. After this he was removed to the Borough Asylum, and for the further notes I am indebted to the kindness of Mr. Evan Powell. The notes say: "He was admitted on Sept. 15th, 1886, with unmistakable signs of general paralysis. He was emotional, and occasionally burst out into tears without apparent cause. He had total loss of sensation in both feet and legs up to the middle of the thighs

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[1] D'Abundo, 'Paralisi Generale Progressiva,' Napoli, 1888; and 'London Medical Recorder,' 1888.

Sensation was also lost in both hands and arms, nearly up to the shoulders, where he could only just feel the prick of a pin. The mouth was drawn to the left: there was ptosis of the left eyelid, and tremor in the facial and lingual muscles. *Sept. 26th.*—Epileptiform fit. *Oct. 24th.*—Hallucinations of sight and hearing. Paralysis of the soft palate causes much difficulty of swallowing. *May 9th*, 1887.—Has delusions that people he cannot see are pulling at his arms and legs. He died on July 4th, after being in the asylum 10 months.

Shortly after this case, I was examining the larynx of an epileptic woman, and as soon as she opened wide her mouth and threw back her head, she had an epileptoid attack, much resembling the one described in the earlier part of this paper. There was marked strabismus, at first conjugate to the left, and then convergent; the tongue was moved rapidly from side to side, and respiration ceased, and she became partially unconscious. She attempted to vomit, and then slowly recovered. The whole attack lasted two or three minutes. Subsequent attempts to examine the throat produced slighter attacks, more of a cataleptic nature. Eventually, by making her breathe deeply and rapidly, a good view of the cords was obtained, and a paralysis of the left cord was found. This explained a condition of partial aphonia which had come on suddenly a few days previously. The paralysis passed off in a short time, and its cause was not discovered.

The phenomena in these two cases were very similar. They appear to have been epileptiform attacks, originating in irritation of the laryngeal nerves. Long ago it was shown experimentally in the lower animals that irritation of a sensory nerve could produce an epileptic seizure. It is said that cases which appear to commence as ataxy, and change to general paralysis, should be looked upon as examples of the co-existence of the two affections, and the dominance of the symptoms of one or the other. As this patient had well-marked ataxy four and a half years before his death, it is difficult to suppose he had general paralysis at that time, as the course of the disease in that case would have been very slow; whereas when once mental symptoms were developed, death followed within a year, indicating a rather rapid course. When first seen, a year and a half before his death, he had no mental alienation whatever, except some depression, which was not unnatural in his unfortunate condition. The only signs at all suggestive of general paralysis were the epileptoid attacks, the inequality of the pupils, the facial paralysis, and the thickness of speech. The first was in some measure accounted for by heredity, as his father was an insane epileptic. Inequality of the pupils is occasionally found in locomotor ataxy. The thickness of speech was sufficiently accounted for by the paralysis, and the impaired sensation in the tongue and lips. The characteristic tremor of the lips and tongue was absent at this time. I should mention that he early had some mental depression, but that did not at first suggest to me the oncoming of general paralysis; for I have long been accustomed to associate some appreciable degree of mental change with the majority of cases of chronic cord degeneration. It is natural that a disabling disease, not necessarily associated with much or any pain, should lead to a despondent view of things. But the mental aspect of patients with cord-degenerations is very different from that of the most hopeless, and, perhaps, equally painless, but always cheerful, cases of phthisis; and equally different from the sad resignation of malignant disease. The mental aspect in cases of cord degenerations has seemed to me to tend towards melancholia, with discontent and quarrelsome dissatisfaction. Though tabes dorsalis is said to commence most frequently in the lumbar region of the cord, the early occurrence of ptosis and of atrophy of the disc, indicates an implication of the cranial nerves, or medulla and basal ganglia, or all three in the degenerative process. It is reasonable therefore to infer, that of the same, or similar, character to the system degeneration in the cord, there is a degradation of that perfect state of nutrition of the cerebrum as a whole (we do not know what portion, if any, is the organ of mind), which is necessary for "*mens sana in corpore sano*," though it may not proceed so far as to be readily demonstrable by the microscope.

A CASE OF CEREBRAL TUMOUR, APPARENTLY THE DIRECT RESULT OF A CRANIAL INJURY.

BY WILLIAM DUDLEY, M.B.,

Assistant Medical Officer, West Riding Asylum.

For the history of this case up to the date of the patient's leaving the National Hospital, I am indebted to Dr. Ferrier, who very kindly lent me his notes, and gave me permission to use them.

Frank H——p, formerly a clerk, was serving in the Cape Mounted Rifles. In July, 1886, he was assaulted by three natives, and immediately lost consciousness upon being struck on the head with a stick. He remained unconscious about twelve hours, and during that time was seen by a medical man, who sewed up the wound. On recovery of consciousness he felt very weak, which he attributes to having lost much blood; but he had no paralysis anywhere, and felt only very slight headache. Three days after, whilst reading a novel, he had his first fit, which came on suddenly, without warning, and rendered him at once unconscious. He was told that he jumped up, screamed, got out of bed and struggled. He was put back to bed, and thinks he remained unconscious about an hour.

After the first fit, he had no more the same day; but the next day he had seven or eight. These differed from the first in the fact that he had a warning. A feeling as of a galvanic shock began in the right shoulder, ran down the right side, up again and down the right arm, up the arm again and two or three times up and down the right side and leg, and then, reaching the right side of the neck and head, he lost consciousness. When unconscious he believes he was stiff in his limbs, but did not struggle or cry out.

For a week he had four or five fits daily of this character, and during this time he suffered from headache which seemed to radiate from a point about two inches above the left ear and round to the other side of the head. After the first week of fits, the warning sensation used to begin in the right arm, but he cannot localise the place of beginning. He says the arm felt as though running away from him, and he would put out the other to grab it, but never could. The sensation would spread to the lip and tongue, but since the first week he has never felt it in the leg. He then lost consciousness, and the spasms, he was told, passed down the right side and were confined to it. He had these fits for about three months, about twelve a day, coming on more often in the daytime, but occasionally in his sleep. The night fits were more severe than the day ones, and he sometimes became unconscious without the warning sensation.

Just after fits, for a few minutes, he used to have thickness and difficulty in speech, but recovered again. He also had at this time a little weakness of the right arm, and used to limp a little. He had no vomiting at any time.

The original wound healed about two months after the accident.

In February, 1887, he underwent an operation (trephining). For a time he was much improved. He felt quite well. He walked without limping, and had no fits for six weeks. Then he had another fit similar to the last described, which was shortly followed by others. On June 1st he started for England. Just on starting he had eight fits, and then completely lost power of speech, but had no marked paralysis. He understood what people said, and knew what he wanted to say, but could not get it out. He could walk about shortly after, but was slow in recovering speech, which has gradually improved to the present time (July 1st, 1887). During the voyage he had a great many fits of the same character as before. He arrived in England on the 18th of June. On the 28th he had three fits, two being severe and one slight. Since starting home he has been slightly lame again, and he has been a little deaf lately.

On July 1st, 1887, patient was admitted into the National Hospital for the paralysed and epileptic.

On admission.—Has slight weakness of right leg. Does not raise the toes well from the ground. There is weakness of lower part of right side of face.

There are two minute vascular dilatations on the outer side of right optic disc. The retinal vessels with which they are connected are not visible. No evidence of past neuritis.

Senses of smell and taste do not seem normally acute. He calls bitters sour.

He says there is one spot always tender, and pain persists for hours after it has been pressed on. The spot is exactly in mid-line, seven inches from root of nose.

Six inches from root of nose and one inch to right of median line is a depressed cicatrix from the former operation. On the left side is a depression in the skull at the anterior inferior angle of the parietal bone. There is some tenderness over the left parietal.

July 6th.—At 11 A.M. complained of a "sensation." For about a minute nothing particular was noticed, but patient seemed unable to articulate, and sat up in a constrained attitude, turning slightly to the right. He then sat for half a minute intently watching his right arm, and had his left hand open and in position to catch hold of right. The right arm was then somewhat rigid. At this time he lost consciousness. Directly after this, face turned to right, and violent twitching began at right corner of mouth, and extended over that side of face. There was extreme conjugate deviation of eyes to right, the pupils at first being hidden behind external canthus; when seen they were dilated. While the face was twitched the limbs were quite flaccid at first. but shortly after tonic, spasm first attacked the right arm, which became pronated and turned in. Then the convulsion came on in the left arm and leg and the face was turned over to left, and violent clonic movements began on that side, and then the eyes deviated to the left. Clonic convulsions were then general.

When the convulsion first began, patient turned very livid; but during the premonitory sensation there was no apparent change.

The pupils quickly regained their normal size after the convulsion ceased, and the patient regained consciousness. There was ankle-clonus on both sides for a few seconds after the end of the fit. Knee-jerks not remarkably active. No marked loss of power on right side.

Patient had several fits similar to the last described. Sometimes he experienced a "sensation," without a convulsion.

Shortly after "sensations" or fits, there was distinct loss of tactile sensibility all down the right side, including the right side of face, and right half of tongue. He could taste bitters and sweets on the left side of tongue, not on the right. When some time had elapsed without a fit or "sensation," these symptoms could scarcely be detected.

On September 13th, patient left the hospital in opposition to the advice of Dr. Ferrier, who was contemplating an operation. He came to his brother in Wakefield, and almost at once went to Leeds Infirmary, in which he stayed a fortnight. Then he spent a fortnight with his brother. On October 15th he had three fits; on 16th, one.

According to his brother, he has always had excellent health, apart from the injury, and has always been temperate.

Neuroses in the family were denied.

Patient was admitted into the West Riding Asylum, Oct. 17th, 1887. He recognised his progressing mental failure, and was very anxious that an operation should be performed on him. The risks and the possible results had been previously explained to him.

Oct. 17*th.*—Patient complains by pantomime of frontal headache. When asked to write down what is wrong, he writes "head" pretty distinctly, then he makes some unintelligible marks, and after obvious effort fails to get any farther. He recognises his failure, and is pained by it. When "aches" is written for him he nods assent. He understands all that is said, and occasionally can give an answer of a single word; he makes many attempts to speak, but most of his words are unintelligible. It is observed that the sounds have a more or less distant resemblance to the words he wishes to speak. He explains sufficiently that he can read and understand printed characters, but he fails almost completely to articulate them. Generally the first word or two has some resemblance to the correct one; then there is a confused jumble of sounds in which no resemblance to the correct sounds can be distinguished. He names letters correctly sometimes, but more often fails. If the wrong name is suggested he knows that it is wrong. His attempts at reading are obviously attended by great effort. He names most ordinary objects pretty correctly, but sometimes fails. So far as can be tested, his memory, except for words, appears good.

He does not know when a fit is coming on, but he is able to explain, by pantomime, the features of the fits before unconsciousness supervenes. The right side of the mouth is first drawn down, then the right arm becomes rigid, strongly supinated in the extended position; after this the right leg becomes affected, being extended and rigid. A feeling gradually extends from the leg up the right side to the top of the head (sometimes being painful). At this stage of the description he allows his head to fall back, shuts his eyes, and signifies by a motion of the hand that he becomes unconscious. In some fits, he explains, the right hand is pronated, with the fingers and thumb approximated at the tips into the form of a cone. The hand is never clenched. Synchronously with the spasm of the hand, he sometimes sees a blue light (apparently with both eyes) which appears to move away from him. After a fit his sight is not good, but he can give no account which would lead one to suppose that there is any hemianopsia.

Always during the fit he hears a humming sound like a bee, which he localises near the right ear. He notices no perversion of taste or smell. After a fit his right arm and leg generally feel weaker than those of the opposite side, and the foot feels numb and dead.

Tongue points slightly to the right, the right side of face is distinctly flattened, and naso-labial fold less marked; this is most distinctly seen when he smiles. The mouth is drawn somewhat to the left. No ptosis, no paralysis of ocular muscles. Knee-jerks about equal, normal. Plantar reflex less marked on the right side.

A week after admission he was put upon bromide on account of the distressing headache, and the medicine was continued about three weeks. Great relief followed this treatment and he became more cheerful and brighter; otherwise there was practically no change in his symptoms.

Dec. 26th.—The first fit since admission occurred. He was sitting in an arm-chair. Spasm commenced simultaneously in right arm and leg. The left arm and hand were kept across the chest, there was slight movement in the fingers only of this hand. Eyes directed to the right. Tongue not bitten. The convulsion lasted only two or three seconds, and he recovered consciousness almost immediately, but was more stupid than usual for a time. Three-quarters of an hour afterwards the eyes turned to the right and patient lost the use of the right arm; it seemed to fall dead to his side, but he quickly regained power in it.

Dec. 27th.—Seems as bright as usual. Says "Good morning" distinctly; asked to name a key, says "No, don't know;" he names a hat; calls a knife a "key," but immediately recognises the mistake. He names a watch, but fails to name the chain; calls a button "but." He cannot name a ring, and does not recognise the name when told it repeatedly. These tests evidently cost him great effort. He often appears as if about to speak, and then checks himself suddenly. He often closes his eyes as people do when trying hard to think. Sometimes he shakes his head and smiles, recognising his failure; if his first effort fails he is seldom successful after.

Dec. 29th.—Yesterday patient began to be drowsy and took little heed when addressed. To-day he is almost comatose, and has taken very little food. The pupils are dilated and inactive.

Stertorous breathing set in, and he died on Dec. 30th. Patient did not vomit while in the asylum, and during the last month he seldom complained of headache.

Autopsy.—The skull cap is less capacious in front on the left side, owing to a slight projection inwards of the left frontal bone. In the coronal suture ¾in. to the right of the median line is a circular opening the size of a shilling, the result of a trephine. The margins are smooth and slightly thickened. The aperture is closed externally by the pericranium and internally by the dura mater; the latter is morbidly adherent for some little distance around the opening, and here it projects pouch-like above the general level so as to fill up the aperture in the bone.

On removal of the dura, the brain protrudes and shows marked evidence of increased intra-cranial pressure, the convolutions being flattened. The anterior border of the left hemisphere is somewhat concave, partly from the corresponding convexity in the bone, and partly from an undue prominence at its outer part. The left hemisphere is larger than the right; length, left 8½in., right 7¾in. Greatest width of orbital surface, left 3½in., right 2½in.; greatest width of hemispheres in temporo-sphenoidal regions, left 3⅞in., right 3⅜in.

No morbid appearance is observed in the right hemisphere beyond the evidence of general pressure, and the projection into the pouch of dura mater of a part normal in appearance, of the anterior portion of the ascending frontal.

In the left hemisphere the third frontal and the lower part of the ascending frontal convolutions are much increased in size and altered in contour. The latter measures from side to side 1¼in., and in depth 1⅜in. The surface of both has a glistening, gelatiniform appearance, and the pia cannot be removed. The two posterior gyri of the insula also are much increased in bulk; they are pushed prominently outwards and backwards and feel very hard.

On section of the left hemisphere, a tumour is found occupying the posterior part of the frontal lobe. It is of a somewhat conoid shape with the base upwards, where it measures 2in. transversely and 1½in. from before back. Traced downwards, it passes with an inclination outwards through the external capsule into the operculum, and the two posterior gyri of the Island of Reil, and from these a hardness, gradually vanishing, is traceable into the temporo-sphenoidal lobe. To palpation the tumour seems circumscribed, but it is seen to diffuse itself into the brain matter around. The lenticular and caudate nuclei are thrust inwards upon the lateral ventricle, the cavity of which is thereby almost obliterated, but the posterior corner of the left, the third ventricle, and the right lateral are dilated. The internal capsule shows no naked-eye changes.

For the most part the tumour is of a greyish-white colour, but marked by numerous small bloodvessels. In parts the vessels are numerous enough to give it a rusty red colour, other parts are darker, mottled, or speckled by minute extravasations. In the centre is a focus of softening, the size of a small pea, surrounded by pigmentation. It is easily sliced; in the opercular gyri and at the periphery softer than elsewhere, almost gelatiniform, and of a slaty purple tinge. The two posterior gyri of the insula, on the contrary, are very hard, almost cartilaginous in feel, and of a dead white colour. The grey cortex is almost invisible, being indicated only by a very faint boundary-line of darker shade.

Elsewhere nothing abnormal is found. Microscopically the tumour presents the characters of the soft glioma.

The case had special interest during life from the question arising of the possibility of affording relief by surgical interference. In a healthy man with a good family history, symptoms of irritation of a localised area of cortex followed closely upon a cranial injury, and were attended by slowly advancing symptoms of paralysis pointing to the same region. When the patient first came under accurate observation, he had slight weakness of the right side of face and right leg, he was subject to convulsive seizures, preceded by a warning sensation in the right hand; the seizures began by turning of face to the right, twitching of right corner of mouth, deviation of eyes to the right, and dilatation of pupils; afterwards, spasm attacked the right arm, and finally became general. Some time previously he had lost the power of speech, but now had partly regained it. All this pointed to the third frontal, the posterior part of second frontal, and the middle of the ascending frontal, of the left hemisphere, as being the probable primary site of the lesion.

The nature of the lesion was by no means clear; but the rapidity with which the symptoms followed the injury seemed to me to exclude the idea of a new growth. The sudden supervention of coma prevented an operation, which the symptoms appeared distinctly to call for, and the autopsy disclosed a condition which could not have been benefited by operation.

A CASE OF TABETIC ARTHROPATHY.

BY F. ST. JOHN BULLEN,

Pathologist to the West Riding Asylum.

I. M., æt. 42, pattern-maker, married, was admitted into the West Riding Asylum on the 30th of October, 1888.

Patient's mental symptoms dated from one month before admission. The facts certified regarding him were: "Excited and wild in manner, restless, attempted to injure both his wife and mother, destroyed his furniture, said that he had invented a pattern which would yield him £12,000."

No predisposing element appears in the family history. Patient is stated to have been intemperate.

Mental State.—When examined the day subsequent to his admission, appears free from excitement, coherent, and ready of reply. He denies that he has shown violence towards any one, and insists that there is nothing amiss with him, and that the relieving officer must have brought him to this place—a convalescent home for imbeciles—in order to get rid of him. His house is the cleanest and his children the finest in Leeds. He states that he has invented a pattern for a new dress material which will realise to him £12,000, and to its purchaser £300,000. He is not rich now, but will be shortly, when he has sold the pattern. Is now an abstainer, but was a heavy drinker when a young man.

Physical Condition.—Patient is spare and of small stature. Cranium well-formed, and frontal development good. There is exaggeration of both patellar reflexes and of the plantar also. Neither ankle-klonos nor tap-phenomenon are obtained. Percussion wave good, no wasting of limbs, no rigidity, contracture, tremor or choreiform movements of extremities. Four trials with the dynamometer gave the following—$\frac{27}{31}$: $\frac{24}{30}$: $\frac{24}{30}$: with swing $\frac{32}{32}$. Equilibrium is well maintained, locomotion steady and orderly, co-ordination generally good. There is some feebleness of the lumbar muscles.

Sensibility is good in answer to tests by tickling, pricking, and of temperature; referred sensations are always accurate. The eyes are placed rather widely apart. Irides greyish-blue, no adhesions of iris. Pupils pinhole $\frac{1 \cdot 5}{1 \cdot 7}$ mm.; are fixed to light, accommodatory movements are present in both, but restricted.

Consensual reflex absent in both eyes; reaction to cutaneous stimuli as well as that with volitional movements abolished. Ocular media clear. Visual acuity $= \frac{5 \cdot 5}{20}$ metres. Visual field extensive and equal in both eyes. Colour tests applied, he fails only with blue. Movements of eyeballs perfect in all planes. Hearing is acute and equal both sides. Smell and taste are alike acute and discriminative. The tongue is steady, free from fibrillar tremor, ataxia, divergence or hemi-atrophy. Articulation is good. Skin is free from any hophoneuroses.

There is considerable swelling of the left leg, without much tension, and without any sign of inflammation. No evidence of thrombosis or other obvious cause of the condition exists.

Progress of Case.—The mental symptoms were variable; at one time denying his previous statements regarding the wealth to be derived from his invention, at another he would discuss his plans concerning it, and intimate that he still expected to derive great pecuniary advantages from it. Being importunate and irrational on the subject of returning home, he made his escape over the wall a fortnight after admission, but was soon recaptured. He gradually, however, became less optimistic and restless, more industrious, and improved in bodily health. Was wont to become emotional when questioned; his face had a rather heavy, expressionless aspect, the lips, especially the upper, being held very stiffly, almost immobile, during speech; articulation, however, was distinct. The tongue was slightly tremulous when protruded, and there was marked clonos of the eyelids when patient attempted to keep them firmly closed. Occasionally he had attacks of vomiting, to all appearance independent of gastric irritation and unaccompanied by pain. He stated that for two years previous to his admission into the asylum, he experienced pains shooting down the anterior surface of both thighs, very stinging and severe, which used to rouse him from sleep. Four months after admission he was placed in bed on account of a painless, chronic-like effusion into his left knee-joint. He became at this time once again restless, troublesome and unreasonable, saying "his leg would get well if he was away from here," and threatening to escape on the first opportunity. Stated that he had sold his pattern; did not know how much for, his wife was managing the transaction; wanted three suits of clothes that afternoon; had discovered that he possessed a splendid tenor voice, and meant to make a lot of money by it. He favoured his listeners with a rendering of "My pretty Jane," in a voice akin to that of a street Punch. There was at this time considerable tremor of lips and voice during ordinary speech; the pitch was also rather high, but patient refused to allow any examination of his throat. Speech rather hesitating and slurred. A month subsequently to the first appearance of the effusion into the knee-joint, the latter was found to present the same characters, although in the interim he had been kept in bed. The swelling, on examination, was partly bony, partly the result of effusion into the synovial sac, not transgressing the limits of the latter. Not the slightest pain, tenderness, redness, or sign of inflammatory action existed. There was free mobility, accompanied with very coarse grating, and to some slight extent lateral movement was allowed. Patient insisted on getting up and walking about, as the joint gave him no trouble, and such appeared to be the case, locomotion being unimpaired except by his holding the leg a little stiffly.

By desire he wrote out a description of the onset of the affection, an abstract of which gave the following: "He was running and had a fall, bruising the right knee, which, however, gave him no further trouble. Nor did he notice anything amiss with the left knee until two or three days after, when he found it swollen to nearly twice the size of its fellow, and fancied he must have put it out of joint. He had no pain whatever in connection with it, although he manipulated it as roughly as possible, but simply a feeling of weakness after resting the limb for some time, also after leaning his weight upon it, he felt the joint give way suddenly at times, this again painlessly. After a month he got the swelling down somewhat, as he thought, by rubbing it and driving the blood on its proper course, but even then it was much larger than the other knee."

The ocular symptoms remained unaltered throughout. An important change in the symptoms was found at this date to be the abolition of the patellar reflex on both sides.

The mental symptoms had improved; he acknowledged his previous excitement, had large ideas of his own abilities, but either had lost, or was too cunning to show, delusion. Was discharged, relieved, about six months from the date of admission.

I had the opportunity of seeing him some weeks afterwards, and noted the following:—

"No trace of patient's former exaltation remains. He is quite rational, shows no pride in his vocal powers, nor has he any hope of making his fortune by the sale of his pattern. The patellar reflexes are absent. His gait is slightly but perceptibly affected, the feet being wider apart and the heels planted more noisily than in a normal individual. It is, however, difficult to dissociate the peculiarity of gait from that contributed by the knee-swelling. Equilibrium is plainly disturbed; he is unable to stand with feet approximated and eyes closed without considerable oscillation. When made to walk with closed eyes, there is a manifest uncertainty in his progress, and on turning round, a notable instability of balance. He is able to localise the position of his members well, approximating the fingers of both hands, touching his nose, and recognising figures described in the air with his forefinger, with eyelids shut; denies any numb sensation in the soles of his feet; describes sudden profuse sweatings, preceded by a hot feeling, which have affected him of late, both during day and night, but more especially the latter time.

"Has not had as yet any gastric pain, but once since his discharge a recurrence of the attacks of vomiting. There is now very well-marked fibrillar tremor of face and lips, and to a less extent of tongue. Clonos of eyelids extreme on attempts of the patient to keep them closed. The pupillary anomalies are as before. The knee preserves the same appearance; there is evident bone-grating and distinct lateral mobility, but still no pain."

A CASE OF AMNESIA WITH POST-MORTEM APPEARANCES.

BY F. ST. JOHN BULLEN,

Pathologist to the West Riding Asylum.

S. C., æt. 73. Admitted into the West Riding Asylum on the 29th of November, 1887.

History.—Patient has always been a steady well-conducted woman. Eighteen months ago, she had a fall down nine or ten steps, cutting her face, but not being stunned nor otherwise suffering at the time. Six months subsequent to the accident, paralysis commenced in the right foot and gradually extended upwards to the right arm. The condition of speech at that time not accurately determined. The palsy was never entirely recovered from, and a second stroke seizing her nine weeks previous to her admission rendered the hemiplegia still more complete. Since this latter seizure she is said to have developed delusions, to have become vicious, unmanageable, and depressed.

Family History.—No insanity, apoplexy, &c.

Mental Condition.—There is much dementia, and it is not easy to isolate the patient's amnesic symptoms for this reason. Her memory is very weak, and her answers shew a great inappreciation of facts relating to time and space; relevant as a rule to the enquiry, they are often inaccurate, and she drifts invariably, if allowed, from the subject in hand and becomes incoherent. Understanding more readily simple requirements, she protrudes her tongue when told, gives her hand, and squeezes with it as desired, but does not discriminate between right and left member.

There is no doubt that she hears quite well what is said to her, although she does not always grasp its meaning. Asked her name, replies either, "I cannot, sir, I do not know them," or gives some Christian and surname, always the same one, but never her own, nor can she be made to give the latter, even as a repetition. She states her age is 24 or 34, that she has been a widow eight years, and that her husband was æt. 28 when he died.

Left to herself she rambles on incoherently, omitting very persistently substantives, except those of very familiar use, but using freely verbs, adverbs, and prepositions. Often substitutes words for those wanting, mostly quite of different meaning, and often devoid of any significance. A third apoplectic seizure occurring a few days after her admission, she remarks, "All this right side, this broken side, is a broken mess." Requested to present her right hand and squeeze with it, says, "I cannot make anything of this, I am very much pained, I am very ill." Again, utters such as the following, "I was very broad when you knew me first; they knew that I had no breach in the earth, then they go out and think that I am bleaching, and I could not understand that I was breaching."

Her speech is, however, by no means always so incoherent as this, and, especially on being aroused suddenly from sleep, talks sensibly and correctly enough for a short time, excepting for the substitution of other words or sounds for the nouns occurring. She is unable to repeat what is said to her; shewn articles whose use she appreciates, and told their names, says, "Yes, yes, I know," impatiently, but is quite unable to repeat the word or anything like it. She is aware of her incorrectness or failure of diction, and states annoyedly, "I cannot make anything of this." This extends, as before said, even to her inability to pronounce her own name. There is no very prominent defect of visual acuity, as apparent by such simple tests as are alone applicable to her reduced mental condition. She recognises common objects, as indicated by the use to which she puts them, although in the majority of instances she is not able to assign to them their names. Asked the colour of a scarlet rug, replies, "Yes, I see, but I cannot sound it with my teeth." She has been able both to read and write, but there is now nearly absolute alexia and agraphia. Given a book of moderately large type, she is unable to pronounce words, even monosyllabic. Pointed out a single letter she can identify others, with fair precision when occurring in proximity, but if many words intervene she misses the letter when next met with. Does not mistake one symbol for another, as a rule, unless bearing some similarity of form, as, R, B, P. Does not recognise her own name when written out and presented to her. Asked to pick out certain letters, without a specimen being pointed out to her, she rarely, perhaps only by chance, chooses the correct one. Shewn a watch at twenty-five minutes to eleven, says, "Twenty-five;" on other occasions gives the hour or the odd minutes correctly; not seldom mistakes eleven for twelve, and becomes hopelessly lost when the dial is held upside down. Can read, generally correctly, the numerals indicating the day of the month at the top of a newspaper, though not the day itself, nor the month; if, however, a succession of figures is placed before her, she becomes quite confused over them, but can identify the first and last. She is unable to write by reason of the hemiplegic condition, and expresses her inability when a pencil and note-book are handed to her, by saying, "I cannot; I could, but this side is all wrong now."

Physical Condition.—Patient's expression is placid, and her countenance but sparsely wrinkled. She is able to frown a little; the articulation is somewhat impaired by absence of teeth and dryness of tongue. Pupils dilated, equal and mobile. There is neither facial nor lingual paralysis. Manifest weakness of the right upper extremity exists; myotatic irritability is a little increased; feeble coarse motility is possible—no contracture.

Patient only possesses little control over the right lower limb; the foot is maintained in a position of partial equino-varus, but not rigidly so; no ankle-klonos. Plantar reflex absent on right side, present on the left; testing of patellar reflexes not satisfactory. Locomotion is feeble and most unsteady; the right limb especially is dragged in walking. Owing to the patient's feeble intelligence, only coarse tests of sensation were applicable, but there was certainly some anæsthesia in the right lower extremity, doubtful in the corresponding upper.

Considerable rigidity of the superficial arteries; the urine contained sugar, no albumen. Throughout patient's habits were cleanly, excepting only the comatose stage preceding the fatal issue, and her behaviour quiet. She died seventeen weeks after admission; almost constantly purged and in a condition of semi-stupor for many days previous to death. Two evenings before the latter occurrence her temperature rose suddenly to 105·8°, falling the next day to normal.

AUTOPSY.

Brain.—Some chronic thickening of the inner membranes with considerable serosity is present, compensatory to more or less general wasting of the cerebrum. Previous to stripping the organ maintains a fairly plump, firm aspect; the arteries at the base shew numerous patches of atheroma, the middle cerebrals being especially diseased; their lumen is in several parts nearly occluded; on both sides they are distributed to the occipital region. There is recent blood film over the vertices of the left central gyri; the median end of the left ascending parietal is, above all other convolutions, wasted; the ascending frontal of the same side much less so, and not so much as the central gyri of the right hemisphere. The following is to be observed in the left hemisphere:—

There is great wasting of the posterior half of the superior temporo-sphenoidal gyrus, the portion anterior to this being also, though to a less extent, atrophied. The posterior third of the middle temporo-sphenoidal convolution, its continuation into the angular gyrus, and the latter itself, are converted to riband-like tenuity. Yellow softening affects the hinder part of the floor of the Sylvian fissure. The temporal annectant gyrus of the insular is still to be seen, but greatly wasted and softened, whilst the digitation anterior to it is perceptible only as a yellow-stained, slightly-marked ridge; the third and fourth digitations are sunken and widely differing from the plump, bold contour of those of the right insula.

The gyri constituting the operculum are apparently free from wasting.

In the right hemisphere the following parts were involved in yellow softening:—

The middle portion of the second temporo-sphenoidal; also an area the size of a crown-piece, which comprised the ensuing:

Posterior part of angular, second and, in part, third annectant gyri, second occipital and a portion of third, extending to nearly the tip of the occipital lobe. The posterior extremities of the first temporo-sphenoidal and supra-marginal also received extensions of the ramollissement to a slight degree.

On transverse sections of the hemispheres being made, it is found that on the left side, the softening of the gyri extends the whole of their depth as a pulpy mass, whilst a greatly diminished consistence of the white matter of the centrum ovale existed to nearly the posterior horn of the ventricle. Before the latter was opened, the medullary stratum forming its roof was seen to be free from softening, but of a reddish hue from the presence of a number of vessels. The ependyma of the ventricle had a hard, firm feel, and the white matter immediately surrounding the posterior cornu was of remarkable toughness, evidently sclerosed, and forming an effectual barrier between the softened and non-softened parts as a tough leathery wall, standing ridge-like above the neighbouring brain substance of lessened consistence. Beyond this condition of the walls of the cornu, nothing else is noticeable. At the level of section of the upper planes of the caudate nucleus, appear in the white substance two holes, severally the size of a small pea and a large pin's-head; these are the cut orifices of obliquely-placed channellings, opposite at this level, the anterior end of the posterior cornu of the ventricle: the larger opening is external of the two; tracing it towards the base it pursues a course having the direction, backwards, inwards and downwards towards the cornu; following it in the reverse direction, it makes its appearance externally in the lower part of the Rolandic fissure between the two central gyri. The smaller canal, situated internally to the preceding, opens by a small aperture in the cortical substance just anterior to it; traced backwards, it runs a somewhat parallel course and ends sooner in a cœcal excavation. The walls of these channellings are free from discoloration, and the surrounding white matter is but slightly below the consistence of the general medullary mass.

At the summit of the ascending frontal gyrus in the same section-plane is a small patch of softening, involving the deeper layers of the cortex and the subjacent white substance to an extent equal to the sphere of a small pea.

On slicing more deeply, so as to expose the segments of the lenticular nucleus, the capsules, thalamus, &c., it is obvious that the greater part of the pulvinar has undergone softening, so that its texture, though not actually broken down, is gelatiniform and tremulous. The geniculate bodies would appear to have escaped involvement, and the corpora quadrigemina are distinctly free from appreciable lesion, nor is there any perceptible change in the optic tracts. On the other hand, the pineal gland is much wasted; it remains as an irregular nodule, the size of a large pin's head; the peduncles are barely traceable. The posterior part of the outer segment of the lenticular nucleus, the external capsule, claustrum and cortical grey matter are affected by pulpy softening.

As regards the cortex corresponding to the insula, it is practically absent through the posterior half of the latter; further forwards it gradually passes from the linear to the stratified condition. The softening involving the third segment of the lenticular nucleus and the external capsule can be traced downwards, inwards and forwards, gradually diminishing in area, until passing beneath the anterior commissure, it ends in the grey matter of the basal surface.

Though it is questionable whether the posterior portion of the hinder division of the inner capsule can have altogether escaped involvement, yet the anterior bend is undeniably perfectly intact.

In the right hemisphere, very similar appearances as regards inward extension and distribution of softening exist, although the latter commences more posteriorly. From the cortex of the occipital lobe, inwards, the centrum ovale is involved in yellow softening until only a like leathery barrier to that on the left side separates it from the ventricle. In both cases the optic striations are completely severed, but on the right side neither optic thalamus nor insula have undergone change. The operculum like its fellow has also escaped.

There are three small cavities in the third segment of the R. lenticular nucleus and one in the claustrum, its posterior part, slightly involving the surrounding external capsule. From none of these does any tract of softening proceed. The outer third of the pes of left crus shewed degeneration.

NEURITIS OF THE MEDIAN NERVE.

BY WILLIAM L. BUXTON, M.B.,

Assistant Medical Officer, South Yorkshire Asylum, Sheffield.

SEVERAL points of interest, relating especially to the area of distribution of anæsthesia in lesions of the median nerve and the order of return of the various temporarily abolished lesions of sensation are brought out in the following case:—

T. P., on December 10th, 1886, fell heavily on a broken wash-hand basin, inflicting a wound on the front of his right wrist. The wound extended from the ulnar side of the radial artery to just beyond the middle line of the wrist. The tendon of the palmaris longus was divided, but careful examination failed to detect either division of the median nerve or any anæsthesia in its area of distribution. Under appropriate treatment the wound progressed favourably until the fifth or sixth day, when inflammation with slight suppuration in the depth of the wound set in, coincident with the appearance of which pain, described as shooting down the inner side of the thumb and radial side of the index finger, came on, was severe and was always worse at night. A spot exquisitely tender on pressure was also present, at the site of the original wound. The shooting pain gradually became less marked, and at this time the following condition was found:—Complete loss of sensation to touch, pain and temperature in the palm of the hand from rather behind the centre of the metacarpal bone of the thumb inwards as far as a line corresponding to the middle line of the middle finger, and forward as shown in diagrams A and B (p. 520) on to the thumb in front and slightly to the inner side but not extending to the tip, in the index finger involving the radial side only as far as the middle line in front, but scarcely so far behind, and in the middle finger also confined to the radial half. At all parts the anæsthetic faded gradually into the healthy regions as indicated by light shading in the diagram. Gradually the muscles of the ball of the thumb wasted so that the thumb could not be brought over the palm, and none of them ultimately reacted to either galvanism or faradism. The hand was easily affected by cold, and the skin became smooth and glossy, but otherwise healthy. He could not make a good fist.

Progress.—Limitation of the original area of anæsthesia was the first apparent change. Three months after the accident my notes show the following condition, when diagrams C and D were also taken.

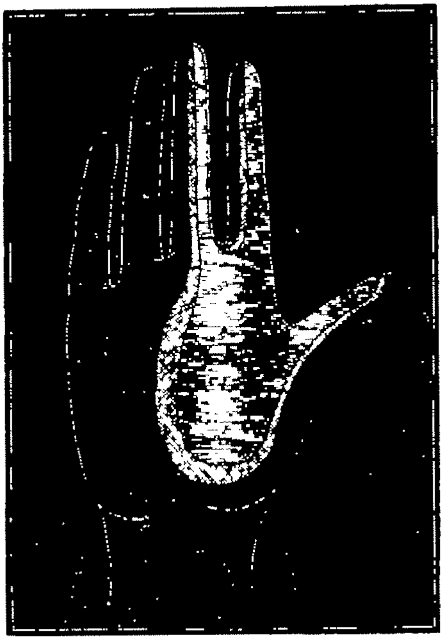
Fig. A.

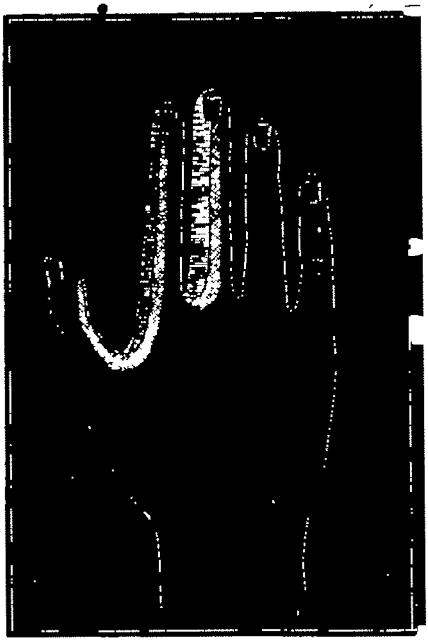
Fig. B.

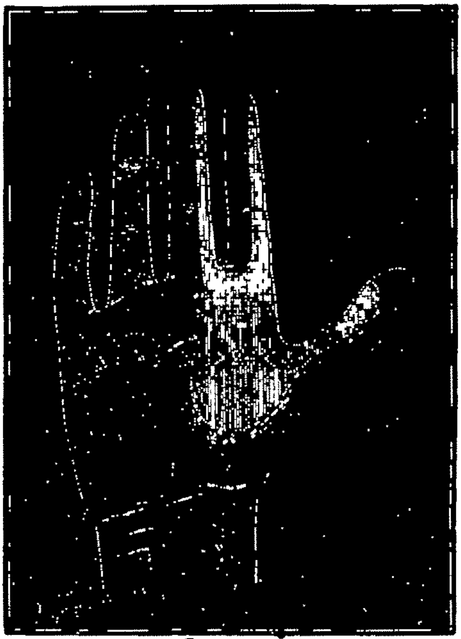
Fig. C

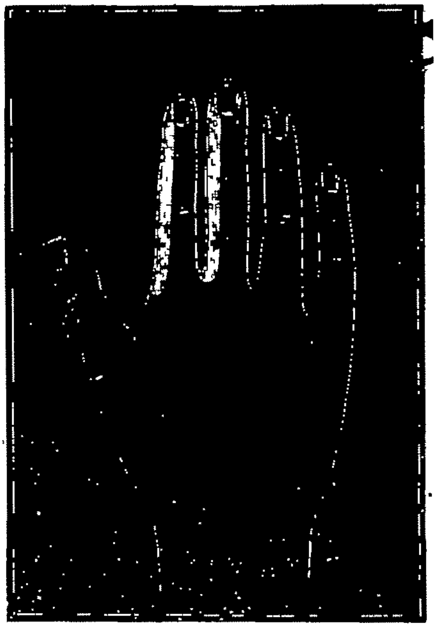
Fig. D.

1. Limitation of the original anæsthetic area. 2. Return of the appreciation of the sense of contact over all the primarily affected parts, though this sense is imperfect, everything being described as "woolley" or "gloved," and points being distinguished as two only in the palm at 24 mm. apart. 3. Return of the appreciation of pain over that area in diagram C indicated by lines in contradistinction to squares, and lastly there is as yet no return of the temperature sense in any part. By this time tumification of the wound had disappeared, and the painful spot was difficult to detect, perhaps absent. So far no voluntary movement of the paralysed muscles had returned. The next note, seven days later, showed that the temperature sense had commenced to return, and that variations of temperature could now be appreciated over the area shaded in diagram E, which shading is left open anteriorly to show that the return progressed from behind forward. The faradic current now brought the short muscles of the thumb into action, and voluntary power commenced to return, so that by the end of April he could bring the thumb across the palm and form a fairly good fist. The farther progress of the case simply showed gradual and ultimately complete return of sensation and motion, so that nine months after the accident, the hand could be freely used even for playing the piano or banjo.

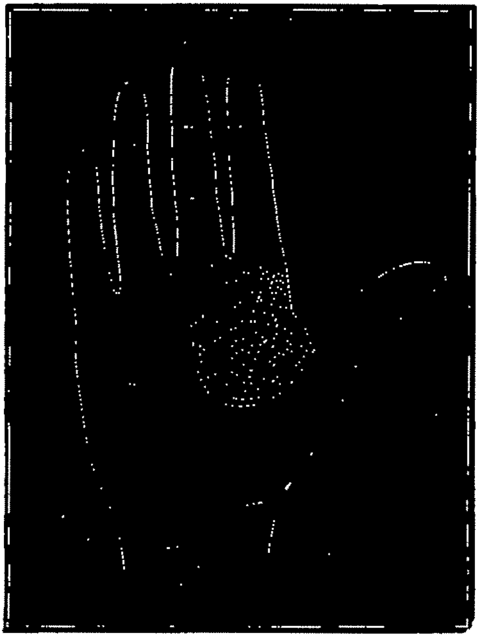

Fig. E.

The treatment adopted throughout was the frequent application of galvanism, and an occasional counter irritant at the site of the injury.

Remarks—The fact that no injury to or anæsthesia in the area of distribution of the median nerve was found immediately after the injury, that coincident with the formation of pus in the wound shooting pain aggravated at night, lasting some time, and paralysis and anæsthesia ultimately supervening, along with the fact that there was an exquisitely tender spot at the site of the original wound, proved clearly that the median nerve was not cut, and that neuritis supervened, ultimately resulting in destruction of the nerve's conductility. Three points are worthy of note. The anæsthetic area was peculiarly distributed; the anæsthesia was absolute, and the order of the returning senses was distinct and definite. With regard to the distribution, Ross[1] concludes that pathological lesions of the median nerve do not corroborate the description of the area of distribution as given in English text-books, but that the description given by Krause and Henle is more in accord; but this case very materially differs from Krause's description, especially, though not by any means entirely, in the fact that the ulnar side of the index finger was not implicated. That the loss of sensation to touch, pain and temperature was absolute, was verified time after time, the patient being under almost daily observation for months. Lastly, improvement followed a distinct and definite course; first, there was limitation of the original area of anæsthesia before any of the senses commenced to return; then came return of the sense of contact, next the appreciation of painful impressions, and one week later the temperature sense returned. In at least the two latter, improvement took place from behind forward, that is advanced from the lesion. The return of the appreciation of simple contact before that of painful impressions seems curious, but probably finds its explanation in the words of Bowlby,[2] that "a more intense impression is necessary to give rise to pain, and that a proportionately perfect nerve supply is requisite, while the sensation of touch can be more or less clearly perceived even when the nerve paths are more or less damaged." It may be asked, was not the nerve only partially destroyed? Evidently it was not even partially divided at the time of accident, and it is difficult to imagine that an acute neuritis could pick out only some bundles of nerve fibre; and even if this was so, the destroyed fibres would not be wholly distributed to the areas found anæsthetic.

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[1] 'BRAIN,' vol. vii., p. 76.

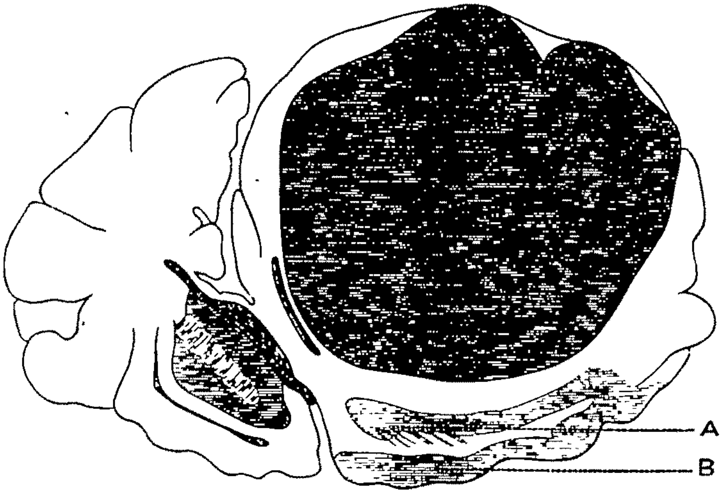
[2] 'Lancet,' June 4, 1887.

young man, but there were no signs of moral perversion in his character. From time to time he was treated by Dr. Sharkey, and subsequently by Dr. Harley, and I am informed by them that the fits were certainly controlled by bromide of potassium.

On February 7th, 1881, he was admitted under Dr. Harley, as he had had six fits in an hour and a half the previous day. For a whole year before that he had been free from attacks.

On June 20th, 1884, he was again admitted into the hospital, suffering from mental aberration after a number of fits which had occurred during the preceding two days. He was vacant in appearance, and would not answer questions. He often picked at the bed-clothes, but was not destructive. Once he got out of bed and ran about the ward. For two or three days a porter was required to watch him. In the state on admission it was noted that the pupils were equal and active to light and during efforts of accommodation; that the tongue was protruded straight; that there was no paralysis; and that the knee-jerks were brisk, but that there was no clonus. There was said to be general hyperæsthesia, with exaggeration of the superficial reflexes. In ten days he was discharged well, so far as his mental condition was concerned.

He was admitted for the third and last time on April 13th, 1887, unconscious and breathing stertorously. The house-physician's description was to the following effect. His face was pallid, and he was sweating freely. The urine was passed into the bed. The pupils were rather small and equal; they did not react to light. The corneal reflex was present, but slight. The arms were somewhat rigid. While the pupils were being examined again they were seen to dilate rapidly under the light, and in two or three seconds he had a tonic spasm, in which the head and eyes deviated to the left. The mouth opened and the left side of it was retracted. At the same time the left arm and leg were raised from the bed and partially flexed. This was succeeded by a slow movement of extension of these limbs. The tonic spasm then became general, and subsequently the clonic stage supervened. The pupils continued to dilate and the eyes to deviate to the left, even after the clonic movements had begun. The neck remained rigid after all movements had ceased, and the head was directed to the left. Within twenty minutes two similar fits occurred. Some hours later he had five more fits of much less intensity. The pupils were dilated, and there was prolonged tonic spasm of the limbs, but no clonic stage. In these attacks the head deviated to the right; the eyes and mouth were not affected. The commencement of one of these seizures was marked by two or three movements of flexion of the right knee. This series of minor fits was followed shortly by an attack identical with that first described. Then ensued ten minor fits. On the morning of the next day (the convulsions having ceased for several hours) he was said to have been partially conscious, and nodded in answer to questions. The attacks soon recurred, consciousness being quite lost during the intervals. In the evening it was noted that he was lying on the left side groaning and passing his hand over the forehead as though in pain. There was no evidence of paralysis. He could not be roused to take notice. He died on the morning of April 15th, after being comatose for fifteen hours. The temperature varied between 99·4° and 102·6°. To the foregoing clinical account I must add a few additional facts. The patient had never, so far as his friends were aware, been subjected to vomiting. He used to stammer, but I have ascertained that this dated from childhood. Dr. Sharkey informs me that he had from time to time examined his eyes and had not discovered any evidence of optic neuritis.



A, B. Caudate and lenticular nuclei displaced and flattened out; with internal capsule between them.

Post-mortem examination made by myself.—Body of a short, well-nourished man. Lividity marked on posterior surface, less evident anteriorly. Rigor mortis general and well marked.

The skull-cap was removed with some difficulty. There was an area of atrophy, two inches and a quarter in diameter, involving the right frontal and parietal bones just behind the coronal suture, and reaching up to the middle line. The outer limit of this atrophied area corresponded to the coronal suture, and here there was a round perforation one-eighth of an inch in diameter. Immediately around this hole the bone was reduced to a mere shell. There was a corresponding degree of atrophy extending in an horizontal direction from the middle line of the frontal bone to the anterior inferior angle of the parietal bone. The frontal bone between these depressions was thickened and unnaturally vascular.

On the outer surface of the right side of the skull-cap there were two bosses, one corresponding to the anterior inferior angle of the parietal bone, the other to the atrophied area and perforation already mentioned.

The surface of the brain was much injected, but there was no lymph or stickiness anywhere. Occupying the white matter of the right hemisphere was a large, firm tumour. About its centre the growth came to the surface, and the dura mater was firmly adherent over it for a diameter of two and a half inches. The tumour bulged considerably to the left and had caused a deep depression on the median aspect of the left frontal lobe.

On making transverse vertical sections the growth was found to have the following dimensions :—

Pre-frontal	Section	2¼	inches	vertically	. . . .	3¼	inches horizontally.
Pediculo-Frontal	„	2½	„	„	. . . .	3	„ „
Frontal	„	1¾	„	„	. . .	1¾	„ „

The growth extended nearly to the level of the tail of the caudate nucleus. The caudate and lenticular nuclei on the right side were displaced and occupied a horizontal position, as shown in the figure. On microscopical examination the tumour was found to be a sarcoma, composed chiefly of spindle cells. The organs of the body were normal.

Remarks.—This case requires little comment. It is possible that the head injury might have had some share in the causation of the morbid growth. Traumatism in my experience is far from uncommon as an antecedent in cerebral tumour, and I am inclined from a survey of several cases to ascribe some importance to injury as a causation. But apart from this, there is good reason for inferring that the growth in this patient was existent in 1877, when he had his first fit. This being so, the tumour must have had a duration of nearly ten years, death occurring in 1887.

The atrophy of the calvarium, as seen post-mortem, also points to the prolonged existence of the mass. It is certainly very remarkable that no symptoms of paralysis, or indeed of any obvious intellectual disorder, should have been evident in this patient. The only definite indication of the brain mischief, looking back on the case, is the occurrence of convulsions, and little information, even had the existence of coarse disease been suspected during life, could have been drawn from them with respect to localisation. It is worthy of notice also that the symptoms resembled very closely ordinary epilepsy, in their occasional occurrence, in the supervention of mental disorder after the convulsions, and also in the amenability of the attacks to treatment by bromide of potassium.

I have since been thankful for the credit of cerebral surgery that the bosses on the skull had not attracted attention during life. It is clear from the condition of things seen at the autopsy that an attempt at removal would have resulted in failure, and possibly in death.

Critical Digest.

BRAIN AND EYE.—THE PUPIL.

BY JAMES ANDERSON.

THE iris has been described specially by Iwanoff as possessing a sphincter and a dilator muscle, the former supplied by the third nerve or common motoroculi, the latter through the cervical sympathetic. This mechanism along with the contraction and dilatation of the vessels of the iris was held to explain the various pupillary states. Of recent years however there has arisen not only scepticism, but actual disbelief in the existence of a dilator muscle, Grünhagen in particular having strongly denied its existence. The question as to the nature of the radiating fibres in the posterior limiting membrane of the iris is admitted by microscopists to be one of much difficulty, but the preponderance of opinion seems to be distinctly against their being muscular. With the doubtful exception of the rabbit, the otter, and birds, a dilator pupillæ must be excluded, and it is clear if this be so that the dilator nerves of the pupil must supply the sphincter and be inhibitory in function. This is the conclusion reached by Gaskell in his recent work on the "Structure, Distribution and Function of Visceral Nerves"[1] where he says he "cannot see the urgent necessity for a special muscular dilator pupillæ; variations in the extent of the contraction or relaxation of the sphincter are quite sufficient to account for all the differences in the size of the pupil, if only the radial fibres of the iris possess, as Grünhagen thinks they do, a sufficient amount of elasticity." He quotes Grünhagen's experiments, showing that the suspended sphincter can be made to elongate by direct stimulation of the muscle itself, and draws a parallel between the iris and the intestine, as to muscular action and nervous supply. In the early part of the above-mentioned work he shows that the circular muscle of the intestine has a double nerve supply, viscero-motor through the accessory and vagus nerves, and a viscero-inhibitory through the abdominal splanchnics. The iris he holds is another example of a muscular structure supplied by two nerves of opposite character, the one motor, the other inhibitory; the motor by the common oculo motor, the inhibitory by the sympathetic trunk passing upward in the neck, which he terms "cervical splanchnic," and he finds that the dilator fibres of it arise in the dog from the anterior root of the second thoracic nerve, an observation agreeing with those of Ferrier and Yeo in the monkey.

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[1] 'Journal of Physiology,' vol. vii., No. 1, p. 38.

It will readily be seen that the above conclusions imply a most important change in our views as to the physiology of the iris, while at the same time observations on the pathology of the iris can be readily translated into the more modern terminology The radical nature of the change is still more manifest when Gaskell's theory as to the real nature of inhibition is understood, that it is an *anabolic* action, diminished activity with repair of function; while the action of motor nerves is *Katabolic*, increased activity with exhaustion of function.

Fuchs has given[1] a careful account of the superficial anatomy of the iris, noting many new and interesting facts regarding it, which have a direct bearing on the above question. The appearances recorded by him can many of them be observed in one's own iris, as Priestley Smith points out by using lateral focal illumination and a concave mirror of very short focus to look into. The hand lens and oblique mirror of an ordinary refraction ophthalmoscope answer admirably. It will be seen that the iris is divided by a small circle into two zones, pupillary and ciliary. The former is crossed by elevated ridges converging toward the pupil and overlapped at its margin by the dark brown edge of the pigment layer. The latter is divided into three zones, the flat, the plicate, and the peripheral, the middle being marked by a series of nearly concentric furrows. The posterior limiting membrane, which must of course be studied in the dead eye, consists of fine radiating fibres showing no nuclei, and certainly, Fuchs believes, not muscular. When the pupil contracts, all the zones on the anterior surface of the iris, including the black border of the pupil, become broader. The sphincter widens, and the slit-like crypts in the neighbourhood of the small circle become longer and narrower. When the pupil dilates, on the other hand, the pigment border narrows or disappears, the crescentic furrows on the anterior surface deepen, and the crypts widen. The posterior limiting membrane is the only part of the iris not thrown into folds when the pupil dilates, and it is therefore presumably the only structure which takes an active share in producing this dilatation. The pigment layer being closely attached to this membrane is drawn back from the margin of the pupil. Were the dilatation caused by the contraction of any tissue lying in front of the posterior limiting membrane, the pigment layer would rather tend to protrude than to disappear.

In his Hunterian Lectures on the Intraocular Muscles of Mammals and Birds,[2] Jessop gives a good summary of the work recently done in this subject with illustrative and control experiments. He adopts Gaskell's view as to the structure and nerve supply of the iris, the short ciliary or myotic nerves derived from the third nerve, through the lenticular ganglion, the long ciliary or mydriatic nerves from the second dorsal nerve, as Ferrier showed in the monkey, and Gaskell has shown in the dog. These long ciliary or mydriatic fibres leave the central nervous system, as Gaskell shows, among the fine medullated fibres of the anterior root of the second dorsal nerve, pass by the cervical sympathetic, through the superior thoracic and the inferior cervical ganglion to the superior cervical ganglion. So far the fibres retain their medullary sheath, but here they lose it just as the abdominal splanchnics lose theirs in the semi-lunar ganglion, with which, and not with the chain of lateral or proximal ganglia, Gaskell considers the superior cervical ganglion to be serially homologous. From the superior cervical ganglion the mydriatic fibres seem to pass through the petrous portion of the temporal bone with the internal carotid artery to the anterior portion of the Gasserian ganglion, thus gaining the nasal nerve, from which they ultimately branch. Jessop notes that the myosis on ablation of the superior cervical ganglion is greater than on mere section of the cervical sympathetic below it.

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[1] 'Ophth Rev,' Jan. 1886. p 11

[2] Ibid. vol. vi., pp 125, 159, 215.

The action of drugs on the pupil gives us most valuable information as to the physiology of the pupil, more especially the action of atropine, eserine, and cocaine. Jessop in his third lecture (*loc. cit.*) gives an excellent summary of these results. Atropine paralyses the unstriped muscular fibre of the eye-ball, eserine stimulates it, while cocaine stimulates the endings of nerves that relax the muscles. The combined action of any two of these agrees with these results. It is to be noted that atropine increases the mydriasis produced by section of the third nerve, ablation of the ciliary ganglion or section of the short ciliary nerves.

In a monograph of 79 pp.[1] Heddæus gives an account of the reaction of the pupil to light, its observation, measurement, and clinical signification. He asserts, and we believe with reason, that the observations and records of the state of the pupil, especially on the part of neurologists are extremely imperfect, and this he attributes partly to want of uniformity in the mode of observation, partly to the clumsiness and ambiguity of our nomenclature. In this last relation he recommends the uniform use of the terms "direct" and "consensual," the first to express the movement of the iris caused by variation in the amount of light falling on the retina of the eye in question, the second to express the movement produced in the opposite eye by that variation. The term "immobility of pupil" he would apply only to the pupil which has neither the direct nor consensual reaction to light nor the accommodative reaction. Excluding for the moment the reflex centre, it may be said that this condition of immobility implies disease of the centrifugal pupillary fibres. When the accommodative reaction is absent, the light reaction will almost invariably be lost. For the reaction of the pupil to light he suggests the terms "reflex sensibility" and "reflex insensibility" (Reflexempfindlichkeit, Reflextaubheit). Reflex insensibility of the retina, or what is the same thing if the media are proved clear, reflex insensibility of the eye, implies disease of the centripetal pupillary fibres.

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[1] Abstract, 'Ophth. Rev.,' June 1887, p 164.

The methods recommended by Heddæus for testing the pupillary reflex do not differ essentially from those employed by most careful ophthalmic surgeons, but as there is probably some ground for his reproach in the case of neurologists, we may enumerate them. In the simplest method, the patient stands about four feet from a window, looking out into the distance. The observer covers the left eye with his right hand for five to ten seconds, meanwhile watching the right eye. He now uncovers the left eye, and estimates the consensual reaction of the right eye. Again covering and uncovering the left eye, he observes the direct reaction of the left eye. He repeats these actions for the right eye. To exclude possible accommodative reaction, he must see that there is no convergence, and also whether the pause or latent period of the reflex, about 0·5 second, takes place. The pupil contracts to its minimum size with a jerk, widens slightly, and ultimately settles to its final size. If the reaction by this method be doubtful, Heddæus next partly covers the right eye until he can just see the outline of the pupil. He at the same time completely covers the left eye, and keeps both eyes thus covered for about a minute. Now uncovering the left eye, he watches the consensual reaction of the right eye and the direct reaction of the left. If the reaction is still doubtful, he sends the patient to the dark room for a quarter of an hour, the second eye is completely covered or bandaged, and the first is then suddenly exposed to the full daylight. For testing the reaction of different parts of the retina, as in hemianopsia, Heddæus employs the dark room and lamplight reflected into the eye with the ophthalmoscope. He has not satisfied himself of any difference in the reaction of the two halves of the retina in such cases.

The last part of Heddæus' monograph is occupied with an enquiry as to the course of the centripetal pupillary fibres, and he gives reasons for believing that all the fibres of the optic nerve do not serve for vision, but are some of them concerned in reflex action. Stilling and Gudden have asserted that this is so, and Gudden even states that the two groups of fibres are microscopically distinct, the visual fibres being smaller than the pupillary, and becoming atrophied on destruction of an anterior corpus quadrigeminum, while both the pupillary fibres and the pupillary width and reaction are unaltered. That the pupillary reaction to light may persist in bilateral total blindness has been known for some time, and von Græfe's explanation is generally accepted, that in such cases the cause of the loss of vision lies centrally from the point of divergence of the pupillary fibres. But sometimes the ophthalmoscope shows optic atrophy, and yet reflex sensibility persists. Here comes in the difficulty of diagnosing the degree of atrophy. In such cases it may be supposed that the nerves, although much damaged, are yet able to convey the light stimulus, but that behind the point where the pupillary fibres leave the damage is so great as to destroy vision.

Heddæus considers from clinical evidence that the centripetal pupillary fibres run in the optic nerve with the macular fibres, and are more resistant to pathological processes than the visual. Also he agrees with Bechterew that these fibres probably do not pass into the optic tracts and do not decussate in the chiasma, but pass back behind the chiasma directly into the grey matter surrounding the third ventricle, and so reach the oculo motor nucleus of their own side. From this it would follow that dilatation of the third ventricle would cause dilatation of both pupils, and that unilateral destruction of the grey matter in the wall of the third ventricle would cause reflex insensibility on the same side. In support of his conclusion, that reflex sensibility is confined to the macular region, he points out that in deeply pigmented normal-eyed people illuminations of the peripheral parts of the retina may produce absolutely no pupillary reaction. And again, in unilateral mature cataract, direct illumination with the ophthalmoscope of the macular region shows reflex sensibility as higher in the sound eye, while lateral illumination shows it as higher in the cataractous eye. The result in the latter case he believes to be due to the diffusion caused by the lens opacity. If Heddæus' assertion be correct, it follows that in hemianopia there would be no difference in the pupillary reaction of the blind and the functional halves of the retinæ, and this, in opposition to Wernicke, he asserts to be the case. The observation of these cases is admittedly difficult and liable to many fallacies, but the weight of evidence is on the whole against Heddæus.

Steinach[1] has made some interesting observations on the pupils of vertebrate animals. He divides them into three groups, those having a total decussation of the optic fibres (horse, crocodile, &c.), those having a few non-decussating fibres, supplying, according to Gudden, peripheral parts of the retina (rabbit), and lastly, those having a considerable bundle of non-decussating fibres (man, cat, &c.). The first two groups show only direct pupillary reaction, even although they have partially overlapping visual fields, such as the owl. The third group shows both direct and consensual pupillary reaction. From the results in the first two groups Steinach implies first that the pupillary fibres of the oculo-motor nerves decussate, and second, that there is no intra-cerebral commissure between the centres of these fibres. It is clear from a comparison of the views of Heddæus and Steinach that farther anatomical and physiological observation is required before a conclusion can be reached as to the course of the pupillary fibres.

The observations of Hensen and Voelcker and of Kahler and Pick make it practically certain that the most anterior cells of the oculo-motor nucleus supply the ciliary muscle and the next posterior the sphincter iridis, and these cells are connected with the anterior fibres of the oculo-motor nerves. Westphal has recorded a case of ophthalmoplegia externa with careful anatomical examination of the oculo-motor nucleus,[1] which practically settles this question, and accurately localizes in man the cells supplying the intrinsic muscles of the eye-ball. He shows that they form a median and lateral group lying dorsally from the general oculo-motor nucleus between it and the posterior commissure.

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[1] 'Centralbl. fur Physiologie,' 28 May, 1887, p. 105.

In 'BRAIN,' vol. i., Mr. Jonathan Hutchinson gives three papers on the symptom-significance of different states of the pupil, and since then many important papers have appeared in this journal on the subject. Uhthoff has recently published an important paper on the diagnostic value of absence of pupillary reflex.[2] His results, combined with those of Moeli, Thomsen and Siemerling, which he quotes, show that general paralysis and tabes dorsalis account for something like 83 per cent. of the cases of reflex pupillary immobility, showing the extreme value of this symptom in the diagnosis of these affections. It is present in somewhat more than half the cases of general paralysis, and Erb finds the light reaction absent or slight in something like 85 per cent. of the cases of tabes. Syphilis comes next in frequency as a cause of the loss of pupillary reflex, and in that case it is frequently combined with loss of accommodation, the ophthalmoplegia interna of Hutchinson.

Jonathan Hutchinson, jun., has recently made observations[3] on the pupil symptoms met with after injuries to the head, and he summarises his results as follows :—(1) In most cases of concussion, for a variable time, depending on the severity of the injury, the state of the pupil resembles that met with in ordinary anæsthesia produced by ether, chloroform, &c., *i.e.*, slowness to respond to light without marked myosis or mydriasis. (2) In a small proportion of cases of concussion temporary mydriasis (uni- or bi-lateral) is met with. (3) When inflammatory reaction follows severe bruising of the brain, myosis is the rule. (4) In compression of the brain, from meningeal hæmorrhage, mydriasis on the side of the lesion is met with in at least half the cases, double mydriasis occurring next in order of frequency, myosis being very rare.

Systematic and careful observation of the state of the pupils will undoubtedly in many cases assist materially in the diagnosis not only of nervous affections but of general diseases. An inequality or irregularity of the pupils, abnormal largeness or abnormal smallness of the pupils, should invariably lead to a careful investigation as to the cause. An old iritis may lead to the diagnosis of gout, rheumatism, or syphilis, which would otherwise have escaped us. The investigation of small pupils will save us from a diagnosis of rheumatism where tabes dorsalis is the real disease. Dilated insensitive pupils if recognised may save us from diagnosing glaucoma as dyspepsia, a not-unheard-of mistake, and one which few medical men would forget to their dying day.

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[1] 'Ophth. Rev.,' 1887, p. 230

[2] Ibid. 1886, p. 104

[3] Ibid. vol vi., pp 97, 129, 153.

Reviews and Notices of Books.

The Nervous System and the Mind. A treatise on the Dynamics of the Human Organism.—By CHARLES MERCIER, M.D., Lond. 1888.

THE author of this important work does not require a lengthened introduction to the readers of 'BRAIN,' the pages of which already owe to him many of its ablest contributions. It is well known that Dr. Mercier started upon his professional career as a pupil—we may, indeed, say, a favourite pupil—of Dr. Hughlings-Jackson; and in dedicating this work to his old master, he gives appropriate expression to the admiration he still feels for his teaching and character. The next most potent factor in the moulding of Dr. Mercier's mind is to be found in the philosophical writings of Mr. Herbert Spencer, which he has made the subject of prolonged and profound study. Any reader, then, who is acquainted with the writings of these two eminent men, will be well prepared to enter upon the study of the work before us; but, at the same time, we ungrudgingly acknowledge that Dr. Mercier has pondered upon psychological problems sufficiently long and arduously to have attained to the character of an original and independent thinker in his own right.

The work is divided into three parts, comprising the study of (1) nervous processes and physiological actions; (2) the psychological actions which eventuate in conduct; and (3) mind.

In the *first* part of the work the author deals, in the two opening chapters, with such physical problems as the nervous discharge and the nervous resistance. After showing the dependence of the nervous discharge upon the molecular complexity of the grey matter, with its *latent* energy ready to become *active* on the application of external forces, he enumerates the inductive laws of the discharge, giving, at the same time, a deductive interpretation of them in terms of molecular energy. The strength of the discharge, other things being equal, is directly proportional to the strength of the disturbing force or stimulus. Once began, it tends to become stronger and to spread; and its augmentation may be manifested either by an extension of the area of its action, or by an increase in its intensity. What now becomes of it? It finds a vent partly along accustomed and permeable channels, and is then expended chiefly in setting up muscular action, and partly along unaccustomed and more or less impermeable channels, and is then expended in overcoming the resistance, and thus in developing new structure. The author believes that powerful currents escape from the nerve fibres into the ground substance; and if this be true, the molecular disturbance of this substance, which is most probably a form of connective tissue, is the correlative of the highest forms of consciousness. We do not believe that there is any justification whatever for this opinion, and think it more likely that the discharge is expended in increasing the permeability of the fibres which are already partially open, in lengthening the fibres which terminate in blind extremities, so that they come by-and-bye to form new connections with the fibres of adjoining cells, and in the formation of quite new cells and fibres. The author now shows that although the accumulation of energy in the grey matter is a more or less continuous process, and the resistance is a constant factor, yet when the nervous energy is being actively expended, it discharges itself in intermittent shocks; and he makes excellent use of this hypothesis in the explanation of the phenomena of tetanus, tremor, and clonic spasm.

In the physiological part of the first division, Dr. Mercier discusses the co-ordination of muscular contractions which constitute *movements*, and of the series of simultaneous and successive movements which constitute *definite acts*. *Co-ordination* is defined as combination in determinate ratio, and a *movement* as any set of co-ordinated muscular actions. Thus the term movement is made to include the muscular actions which maintain the fixed attitudes of the body as well as those which produce active changes of parts of the body in space. Viewed in regard to the region of the body concerned, movements may be divided into *central* and *peripheral*. The central movements occur in the trunk, and are continuous or tonic, indefinite, few in number, simple in character, and give rise to a coarse or general adjustment of the body; while the peripheral occur in the digits, mouth, and eyes, and are intermittent or clonic, definite, multiple in number, diversified in character, and give rise to adjustments which are very precise and specially adapted to particular ends. Passing now to the consideration of the higher co-ordination, by means of which a group of movements is arranged into a definite act, Dr. Mercier points out that the individual movements are either *simultaneous*, chiefly effected by the central muscles; or *successive*, chiefly effected by the peripheral muscles. The successive movements are of two kinds—*radial* and *lateral*. In the radial, the contractions begin in the central and spread to the peripheral muscles, or begin in the peripheral and spread to the central, these being respectively named *centrifugal* and *centripetal* radial movements. In the lateral, the succession is effected by the peripheral muscles, and the movement spreads from one physiological series to another, as from the arm to the leg, or from one digit to another. After some very suggestive remarks on inhibition, which the exhaustive discussion of the author's views in the last number of 'BRAIN' relieves us from criticising, and on the constitution of nervous centres in general, this part of the work is brought to a close by the enunciation of Mr. Herbert Spencer's theory that the cerebellum is the great centre for the co-ordination of simultaneous, and the cerebrum for the co-ordination of successive movements; the former being the organ of compound co-ordination in space, and the latter the organ of compound co-ordination in time.

In the *second* division of the work, the author enters upon the study of the actions of the body, not as hitherto in relation to one another and to the body itself, but as they effect an adjustment of the organism as a whole to the whole of its surroundings or to its environment. This adjustment is effected neither by the co-ordination of muscular contractions which constitute a movement, nor by that highest co-ordination of movements which constitute a definite act, but by a highest co-ordination of definite acts to a common end, which constitutes *conduct.* It is by the observation of conduct alone that we can judge of the degree of intelligence of others; and in this study, which forms the subject-matter of *objective* psychology, no cognisance is taken of consciousness. The degree of intelligence is estimated by the novelty, the elaborateness, and the precision of the adjustments, and still more by observing whether the adjustments are or are not adapted to self-conservation, the rearing of offspring, and the welfare of the community. Excellent remarks follow on the nervous mechanism which underlies conduct and renders it possible.

In the third and probably most important division of the work, Dr. Mercier deals primarily with states of consciousness, these forming the subject-matter of *subjective* psychology. States of consciousness may be viewed wholly from an introspective standpoint, reference being made solely to their comparison with one another; or they may be viewed in connection with the circumstances in the environment with which they correspond. From the introspective standpoint, states of consciousness may be resolved into feelings, and the relation between feelings. So long as consciousness is occupied with a sound, for example, a simple feeling is experienced; but when the sound changes by becoming suddenly louder or shriller, the feelings of difference and sequence constituting the relations between the two sounds occupy consciousness, a judgment is formed, and the feeling has given place to a cognition or thought. But the primary division of states of consciousness into feelings and thoughts may be cut at right angles by a second introspective division of them into *vivid* and *faint* states. Vivid states are caused by objects present to the senses, and they are consequently named *presentative* feelings and thoughts; while faint states are revivals in consciousness of vivid states previously experienced, and are consequently called *representative* feelings and cognitions. In accordance with these divisions, Mr. Herbert Spencer classified states of consciousness into feelings and cognitions, while each of them are subdivided into serial order according to the descending degree of vividness with which they occupy consciousness, the most vivid being termed presentative, and the next in order, presentative-representative,

representative, re-representative, &c. The advantages Mr. Spencer claimed for this classification are that it represents, in a rude way, the order in which the feelings and cognitions are evolved, the presentative being common to all sentient beings, the presentative-representative to most animals and the human infant, the representative to the higher animals and the child, and the re-representative to adult man, while the feelings and cognitions which are still more remote from actual experience are peculiar to the most highly developed of the human race. It is at this point that Dr. Mercier diverges from his master. Without wholly rejecting the introspective classification, he prefers to regard states of consciousness with reference to the circumstances in the environment with which they correspond. From this point of view, a cognition or thought is the process of establishing relations in the organism in correspondence with relations in the environment. In accordance with this view, thoughts are divided into those in which there is (1) the establishment of a new relation, and (2) the revival of a previously established relation. When a new relation is established, the process is termed *reasoning*, and the result a *judgment* or *concept*. When a previously established relation is revived, if both terms of the relation are wholly represented, the process is termed *remembrance*, and the results are either *memories* or *concepts;* and if one or both of the terms of this relation contain presented elements, the process is termed *perception*, and the results, *percepts*.

Turning now to the feelings, they are divided from the introspective standpoint with reference to their development according to their degree of representativeness, quantitatively into intense or voluminous, and qualitatively into pleasures and pains. But without rejecting this classification, Dr. Mercier regards them as states in the organism which correspond with interactions between the organism and the environment. In accordance with this view, he classifies the feelings—

1. According to the way in which the interaction is begun, whether by the environment, by the organism, or by both.

2. According to the position of the respective feelings in the evolutionary scale, the order being from the low kind of feelings and emotions which affect the conservation of the organism, to the progressively higher emotions which affect the perpetuation of the race, the common welfare and the welfare of others, and to the still higher feelings which are neither conservative nor destructive, and those which correspond with relations between interactions.

3 According to the directness or indirectness of the correspondence of the feeling with the interaction, and the degree of remoteness from direct correspondence, the direct feelings being called *sensations;* the indirect, *emotions;* and the remote, *sentiments*. Following out these principles, the author arranges the feelings into six classes, which, with their sub-classes and genera, require for their representation no less than seventeen elaborately constructed tables. Dr. Mercier's analysis, with its accompanying

classification of thought and of the feelings, constitutes by far the most important as it is the most original portion of the work; and it seems to me now, as it did on reading it a few years ago in the pages of 'Mind,' to be incomparably the ablest contribution to subjective psychology since the publication of Mr. Herbert Spencer's great work.

This book forms such a solid and valuable contribution to psychology that we do not feel much in the humour for fault-finding. We will, however, venture to direct the author's attention to a few points in which the work is susceptible of improvement when the occasion arises, as we trust it soon will, for issuing a second edition. In the first place, we think that the introduction ought to be entirely re-written. Neither would the author's reputation lose anything, nor would his arguments be less forcible and convincing, did he write in a more courteous manner or his brother alienists, did he employ less bombastic epithets in his denunciation of the older psychological terminology, and, above all, were he to abstain from garnishing his sentences by the use of Biblical language. Dr. Mercier ought, in our opinion, to be much more sparing than is his wont of arguments of the sledge-hammer order. A sledge-hammer is a very effective weapon for cracking an opponent's skull, provided only his head be first fixed in the line of descent; but if it is not so fixed, the opponent steps aside, and laughingly observes the blow descend harmlessly by his side. As an example of this kind of argument we may quote Dr. Mercier's attempt to show that the proposition "that a causal relation exists between the events of mind and the movements of matter" is *nonsense*, in the literal meaning that it has *no sense*. Did the present writer maintain the truth of this proposition, Dr. Mercier would have no difficulty in cracking his skull, because he would himself make willing admissions respecting the theory of causation which would fix his head in the line of descent. But suppose that Dr. Mercier has to deal with an opponent who, following Hume, Stuart Mill, and Professor Huxley, regards causation as merely implying the existence of a definite order amongst phenomena established by experience, and who denies that there is in any case a community of nature between cause and effect, or a *nexus* which binds the one to the other, what would be the result of his sledge-hammer blow? None whatever. With such an opponent the author would soon find himself involved in a discussion respecting the theory of causation; and although he might still obtain a victory, he would feel himself under the necessity of laying aside his sledge-hammer and taking to the rapier. In other words, the only effective weapons in discussions of this kind are those which require for their use subtility and skill, and they cannot be safely replaced by exhibitions of mere brute strength. It is not maintained that Dr. Mercier is destitute of skill when he has the patience to exercise it; what is asserted is that he is lacking in sympathy with opponents. If they are not full-blown evolutionists, he does not even care to conceal his contempt for them, and he is much too fond of making a show of his strength by attempting to overbear all resistance by one mighty blow.

The next point we notice is the halting interpretation the author gives of the terms "generality" and "speciality" as applied to movements (p. 94). Is any one the wiser for being told that "by a special movement is meant one which serves a special purpose, and that only"? Taken along with its context, some meaning might be read into this proposition; but even then it is far from being definite enough. This is to be regretted, because these terms are much abused in medical writings generally, where they are hardly ever used, as they ought to be, in the sense attaching to them in embryology, and in the classifications by species of botany and zoology. Generality means, according to Mr. Herbert Spencer, "manifestation in numerous cases;" and conversely speciality means manifestation in few cases. The movements of the trunk are met with in all vertebrate animals, and they are consequently very general. The peripheral movements are, avoiding details, peculiar to the higher vertebrates, and are therefore special; some of them are met with in man alone, and consequently they are still more special; while a few of them occur only in the most highly endowed and educated men, and these attain to the highest degree of speciality. The fact that Dr. Mercier has missed the true meaning of the correlative couple—general and special—has prevented him from making such fruitful use as he might have done of the comparative physiology and embryology of these movements. In his discussion of conduct, Mr. Herbert Spencer's classification of human activities might have been usefully introduced. He arranges them into—(1) "those activities which directly minister to self-preservation; (2) those activities which, by securing the necessaries of life, indirectly minister to self-preservation; (3) those activities which have for their end the rearing and discipline of offspring; (4) those activities which are involved in the maintenance of proper social and political relations; and (5) those miscellaneous activities which fill up the leisure part of life, devoted to the gratification of the tastes and feelings" ('Education,' p. 8). This classification, which is a most important one from the standpoint of alienism, would form the correlate, on the side of objective psychology, to Dr. Mercier's main division in the classification of the feelings on the side of subjective psychology.

The last point we shall notice is that, in his criticism of Mr. Herbert Spencer's classification of the feelings, Dr. Mercier is hardly fair. An author has a right to expect that his work should be judged according to the aim he has set himself to accomplish, and not according to an altogether different standard set up for him by his critic. Suppose an engineer, for example, erects a bridge for the purpose of carrying troops across a river, what should we think of a brother engineer who should criticise his construction as if it were intended as a permanent roadway for generations of peaceable citizens? Now the particular purpose for which Mr. Herbert Spencer advanced his classification of the cognations and feelings was to arrange them in their due positions in the evolutionary scale; and the advantages he claims for his classification, which he distinctly states to be a provisional one, are that it is reached without a lengthened inquiry, and that it is "in harmony" with the order disclosed by detailed analysis. But that is not all. Mr. Spencer not only *must* know that in adopting a *serial* instead of a *specific* classification, he is using a very imperfect method, but he also distinctly states against his own classification almost every objection urged by his critic. The objections we have to urge against Dr. Mercier's strictures are that he criticises Mr. Spencer's classification as if it were intended to be a classification by species instead of a serial one; as if it were advanced as the result of a detailed analysis, instead of for its readiness of application; as if it were a completed classification, from which all objection had been removed, instead of being avowedly incomplete, and having most of the objections to it distinctly stated by its author; and as if it were regarded as a permanent classification, to be used for all purposes whatsoever, instead of being a provisional classification used only for a particular purpose. That Mr. Herbert Spencer had contemplated a very different classification of the feelings from this one, may be gathered from his own words. "We may," he says, "study the evolution of the emotions up through the various grades of the animal kingdom, observing which of them are earliest, and exist with the lowest organisation and intelligence; in what order the others accompany higher endowments, and how they are severally related to the conditions of life. In the second place, we may note the emotional differences between the lower and the higher human races—may regard as earlier and simpler those feelings which are common to both, and as later and more compound those which are characteristic of the more civilised. In the third place, we may observe the order in which the emotions unfold during the progress from infancy to maturity. And lastly, comparing these three kinds of emotional development, displayed in the ascending grades of the animal kingdom, in the advance of the civilised races, and in individual history, we may see in what respects they harmonise, and what are the implied general truths" ('Essays,' vol. i., p. 310) This passage requires no comment; and elaborate and valuable as we believe Dr. Mercier's classification of the feelings and emotions to be, it seems to us yet to fall considerably short of the ideal set forth in the above quotation. The great objection we have, indeed, to his classification is that the genetic relations of the higher and more compound to the lower and simpler emotions find only an imperfect expression in it; but we shall look for this defect to be removed in the further development of the author's system. It would be an injustice to Dr. Mercier not to mention that all he claims for his classification is that it is "founded on the principle of evolution, and that it harmonises with Mr. Spencer's psychology, of which it is, indeed, an extensive corollary." This statement is sufficiently modest; but we still think that a good deal of the criticism he directs to Mr. Spencer's classification is misdirected, and might have been spared.

But whatever may be the faults and shortcomings of this book, it cannot be denied that it is a noble contribution to psychology. Its intrinsic merits are great, and it is still greater in promise. For it must be remembered that the present volume only forms an introduction to the author's complete scheme, and that upon it as a foundation he intends to erect a system of scientific alienism.

Several of the distinctive questions of alienism are discussed in this volume with sufficient fulness to enable us to form a tolerably accurate opinion of Dr. Mercier's views with regard to them. He will doubtless object (p. 213) to the distinction usually made between emotional and intellectual insanity. He, however, admits that in normal consciousness the emotions and thoughts, especially in their higher forms, are inextricably blinded. Yet this does not prevent him from separating thought and feeling in his classification of mental states. In other words, he allows the practical utility of this distinction to override all theoretical considerations; and when he comes to deal more specifically with the morbid phenomena of consciousness, he may find the retention of the old division of insanity into moral and intellectual practically advantageous, whatever may be the theoretical objections to it. The author's opinion of the particular disorder of the mind which constitutes insanity, may be gathered from his analysis of thought and his subsequent remarks. When once we pass beyond the purely introspective view of consciousness, thought is the establishment of a mental relation in correspondence with a relation in the environment. In insanity, the mental relations, when considered amongst themselves, may be valid; but their correspondence with external relations is not only unadjusted, but is incapable of being adjusted (p. 250). Here we come within sight of a scientific definition of the terms delusion, illusion and hallucination, which will replace the imperfect and often absurd meanings given to them in current writings. The writer is tempted to reproduce here definitions of these terms which he gave in a course of lectures delivered in the early part of last year. Dividing knowledge into that which is obtained directly through the senses, and that which is *indirectly* established by reasoning, it may be said that hallucinations and illusions are a disorder of the former, and delusions of the latter. *Hallucinations* and *illusions* are *unreal* perceptions, the presented factors in the former being generated in the organism, and of the latter in external circumstances. In insanity there is in addition a conviction of the reality of these perceptions which cannot be corrected by experience. A *delusion* is primarily a belief in a proposition, the truth of which, if challenged, could only be established by a process of reasoning. In the next place the proposition does not correspond to fact, and it is consequently a *false* belief. If, however, strong reasons exist which would give semblance of truth to the proposition, we should hardly call it a delusion, and consequently we must amend our definition by saying that a delusion is a false and *groundless* belief. But before a delusion is reached, another essential constituent must be added, that the belief in the truth of the proposition is incapable of being corrected by experience, and we now reach the ultimate definition of a delusion as a false, groundless, and *incorrigible* belief. We cannot enter here upon a defence of these definitions; but if the terms used be translated, as is easily done, into Spencerian language, it will be found that they do not differ much from Dr. Mercier's views.

We have said that we did not feel much in the humour of fault-finding, and yet have gone on fault-finding until we have become quite censorious. The author will, we trust, believe us when we say that the reason and justification for our fault-finding is to be found in the very excellence of his work. In the volume before us is laid in broad lines the foundations of a system of alienism which, when completed, will, judging from the author's comprehensive grasp of his subject, challenge the admiration of future generations, and will be read and studied when the fleeting publications with which the medical press is teeming are placed on the shelf and forgotten. Dr. Mercier's work is, in short, far too good for a critic to content himself with a simple analysis of its contents, followed by a few platitudes and indiscriminate eulogy. We believe that amongst the younger generation of the profession are to be found a great many earnest, thoughtful, and learned men, to whom this volume will be most welcome, and who will carefully read, re-read, and ponder the detailed psychological analysis, the comprehensive generalisations, and the valuable hypotheses which are so lucidly expressed in its pages.

JAMES ROSS, M.D.

Physiologie des Exercices du Corps. Par le Dr. FERNAND LAGRANGE. 1 vol. pp. 372. Paris. Alcan, 1888.

THIS volume of the "International Scientific Series" is calculated to direct very timely attention to a subject of great importance. Calisthenics are just now the fashion, and a book dealing methodically with the rationale of muscular therapeutics was much wanted, in order that the limits of this mode of treatment may be appreciated by the profession and the public. Dr. Lagrange's work forms part of the "International Scientific Series," and we may confidently anticipate the appearance of an English edition at no distant date. Although the work is presumably written for intelligent readers of all classes, and takes the form of a popular exposition of the subject, we have no hesitation in strongly recommending its perusal to our readers. Dr. Lagrange has an admirable grasp of his subject, and writes so tersely and lucidly, and has so many new ways of presenting old facts, that few can read his work without instruction and none without pleasure.

Dr. Lagrange begins with an excellent account of the physiology of muscle, nerve, spinal cord, and brain in relation to movement. The facts he lays before the reader in these sections are indispensable for obtaining a philosophic grasp of the subject, but need not detain us as they are all certainly familiar to the readers of 'BRAIN.' The author does well, however, to dwell upon the fact that the least movement performed by the human machine sets many of its wheels in motion. "To move the forearm, the arm needs to be fixed, the arm must be steadied on the shoulder, the shoulder on the thorax and spinal column; but these being supported by the pelvis and legs, it follows that the whole body must co-operate to move the forearm." This is a fact too often forgotten, but it needs to be constantly borne in mind when considering the localisation of the so-called cerebral motor centres. Again, it is well to insist that "In performing a movement a muscle never acts without the simultaneous contraction of its antagonising muscle, which serves, as it were, as a counterpoise and check."

"Effort" is defined as that arrest of respiration which takes place during vigorous muscular acts for the purpose of giving support to those muscles which are attached to the thorax. "Effort," however, is often seen in acts where the expenditure of force is on the whole small. It is seen whenever it becomes necessary to contract a group of muscles with all the force of which they are capable. Look at a man cracking walnuts with his fingers. If the fingers be strong and the shell brittle, the muscular contraction is limited to the forearm, the face remains calm and the respiration is undisturbed; the nut is cracked without effort. But if the man has to use all his power to break the shell, the muscular contraction spreads from the forearm to the arm, shoulder, neck, chest, and abdomen; breathing stops, the face reddens, the veins of the neck and face swell, and when the shell at length gives way a kind of groan is heard, like that of the porter who lifts a burden from the ground. Some surprise may be felt that an act involving directly only the flexors of the fingers should necessitate the contraction of such distant muscles, and that in order to squeeze with the hand the abdominal muscles become essential. But the body being composed of many movable pieces, and no muscle being able to put forth all its strength unless one of its ends be rigidly fixed, it follows that for the fixation of the forearm the arm, shoulder, thorax, and pelvis must be steadied. This end cannot be attained unless a great mass of air is in the lung, for the lung distended with air is the only support for the movable bones of the thorax. The distended lung forms a sort of resisting cushion upon which the ribs rest firmly as they are pulled downwards by the abdominal muscles. The extra flow of blood to the contracting muscle is the main factor which produces the increased activity of the circulation which is one of the initial phenomena of muscular exercise, and which produces an increased activity of vital processes in every organ of the body inclusive of the brain. The Peripatetics disputed as they walked about, and J. J. Rousseau has said, "Movement and walking favour the play of the intellect and facilitate the work of thinking" Exercise, indeed, may produce excitement not unlike drunkenness, as is seen in the dancing dervishes, or without going so far, may cause a great flow of animal spirits, as is seen every day in the ball-room.

The fatigue of a muscle, as seen in the laboratory, when contraction no longer follows stimulation, is caused by the paralysing effect of the chemical products of contraction. This form of fatigue probably never occurs in nature except in the case of hunted animals, and when thus caused is sufficient of itself to produce death. The painful sensation of fatigue is due to impressions made upon the intramuscular nerves, and occurs long before the physiological exhaustion of the muscle. It is a sensation which preserves us from the deadly effects which would result from the presence in the blood of an excessive quantity of the chemical products of muscular work. The sensation of fatigue is a cerebral phenomenon, and its occurrence is largely influenced by the amount of cerebral involved by any particular act.

One of the commonest results of active exercise is breathlessness, or as athletes say, getting "blown." This condition is one of considerable interest, and Lagrange deals with it at length under the name of *essoufflement.*

The intensity of breathlessness is in direct proportion to the amount of force expended in a given time. It is a sort of physiological measure of the amount of work. When the amount of work done is very great, as when a man runs up four pairs of stairs, and thus raises 140 lb. through 50 feet in a minute (or let us say 7000 foot-pounds, or more than 3 foot-tons, in a minute), breathlessness is necessarily produced with great quickness. The exercise of a great mass of muscles loads the blood with carbonic acid, and when the production of carbonic acid is greater than the eliminating power of the lung, the respiration gets troubled and the worker is "blown."

Exercises of the arms, with a muscular mass much less than that of the legs, are slow to produce breathlessness, and the arm-worker is apt to get tired before he is "blown." When hard work is done with the legs, however, the reverse holds good, as the runner is often "blown" before he is tired.

If, during exercise, an "effort" as defined above be made, and the respiration be temporarily arrested for the purpose, it follows as a necessary consequence that "loss of wind" is the sooner produced.

Again, physical or emotional irritation producing reflex effects upon respiration, such as the sudden impact of cold, fear, anger, or laughter, by checking the eliminative power of the lung tend to hasten loss of wind. The man who scolds or whips a willing horse so as to produce fear during work effectually defeats his object. There is a striking resemblance between a man in a state of terror and one "blown" by exercise.

In the early stages of exercise, pulse and respiration are alike quickened, and there is a determination of blood to the lung. As a result of this the elimination of carbonic acid is increased, and the effects of exercise are compensated. If the respiration, however, be unduly quickened, the true function of the lung is lessened and carbonic acid accumulates in the blood. The hurrying ventricles of the heart also fail to compensate by rapidity what they lose in force, and thus is brought about a condition of things physiologically similar to asphyxia—passive congestion of the lung, a distended right heart, and a left ventricle tending to increase in feebleness. When the excessive exercise inducing this alarming state ceases, Lagrange has observed that the respirations reach the normal sooner than the pulse, and the following is the mean result of observations made upon himself and two guides during an ascent of Mont Canigou:—

	Pulse.	Respiration.
Complete Repose	62	14
During the most severe climbing	123	30
Five minutes after reaching summit . . .	117	14

According to Lagrange excessive exercise causing breathlessness alters the rhythm of respiration. During repose the times occupied by inspiration and expiration are, as he says, exactly equal, but observations made on a man who has run himself out of breath show that the rhythm has completely altered, inspiration being much longer than expiration. His explanation of this fact is as follows: The first effect of exercise is the quickening of the blood current, and the active congestion of the lungs. The need is felt of disembarrassing the lung, and to this end the aspirating effect of inspiration is brought to play. This gives comfort to the breathless man; but when the blood current slackens during expiration the lung is again engorged, and hence the quick instinctive return to inspiration. The lung has need of disencumbering itself of blood and carbonic acid; the one is favoured by inspiration and the other by expiration, and thus the "blown" athlete is placed upon the horns of a dilemma.

A person completely "blown" is in a critical condition, and many of the deaths which take place on the race-course are due to a fatal syncope supervening on the asphyxia caused by loss of wind. If we are to get good results from exercise, and free from all danger of overstraining the heart and lungs, we must choose such moderate exercises as do not involve an excessive amount of work or the making of "efforts." Loss of wind it need not be said depends, as to the time of its occurrence, very largely upon training and the state of health.

There are other effects of exercise which are not quite so immediate as breathlessness. After unwonted exertion a man is apt to feel stiff and "knocked up," and this condition is described by Lagrange under the name of *courbature*. It is characterised by sleeplessness and restlessness, notwithstanding a feeling of being tired out, and this is accompanied by some heaviness in the head, a coated tongue, loss of appetite, and stiff and painful muscles. Lagrange asserts that exceptionally this condition is accompanied by fever, but he does not give any details as to temperatures, &c. The liability to be knocked up varies with the state of health, and reaches the vanishing point with a man "in training." One of the most common symptoms of this condition is the formation in the urine of a deposit of urates, and the circumstances which favour these deposits have been carefully studied by Lagrange on himself and others.

When a healthy man takes some exceptional exercise the appearance of his urine varies with the time of its secretion, the amount of exercise and the state of training. Lagrange asserts that the effect of exercise on the urine is not immediate, and that it is useless to expect the appearance of urinary sediments earlier than three hours after exercise. Other things being equal, it is asserted that the urinary sediments are directly proportional to the work done by the muscles. "According as the exercise is more or less violent the precipitate may vary, from a slight cloud appearing only once to a thick muddy deposit lasting twenty-four hours." The liability to the precipitation of urates lessens with training until no amount of work seems capable of producing it. But, and this is noteworthy, if a given exercise for which the athlete is in training be discontinued and another be substituted involving the use of different muscles, the liability to *malaise*, stiffness and urinary deposit reappears. "The man accustomed to hard walking feels no trouble after a long tramp, but he will be knocked up by a short bout of fencing if he be unaccustomed to that exercise. An examination of the urine will show that this fluid, although it remain clear after a twelve-hours' walk, becomes cloudy with urates after twenty minutes with the foils." It is almost needless to say that any impairment of health increases the liability to *courbature* and the accompanying urinary sediments. Lagrange claims to have proved that the urinary deposits are not due to a concentration of the urine as a result of heavy sweating. He gives an interesting account of how he rowed with a friend (both being at the time in perfect training) from Limoges to Paimbœuf, near the mouth of the Loire, a distance of 550 kilomètres, with the additional labour of dragging the boat over the locks, eighty-three in number. The work done was very excessive, and the food very substantial, with lots of wine and coffee. Yet they were never knocked up, and the urine, examined every day, never showed any sediment. If these sediments be the result of sweating, surely they should have occurred on Aug. 10, the second day of their journey, when they rowed 50 kilomètres under a broiling sun and slept at night beneath a feather quilt in a room over a baker's oven. A slight mishap to the boat gave them leisure on the following day for repeated urinary examinations, but "in spite of sweating, which had soaked our garments by day and our bedclothes at night, no urinary deposit occurred in my friend or myself."

An analysis made by Mr. Papon of the urine of a man out of training after a long bout of fencing, gave 1·43 grams of uric acid per litre, while the urine of the same person after training, and after the same amount of exercise, gave only 0·60 grams per litre. The *malaise* of fatigue is largely due, according to Lagrange, to a species of uræmia, and the well-known fact, that unwonted exercise often determines an attack of gout, seems to give support to this theory. The fact that training renders the liability to this condition very much less is probably due to the fact that the man out of training has his muscles encumbered with tissues of reserve, which disappear as his training advances.

Just as breathlessness is due to a self-poisoning by carbonic acid, so knocking up from fatigue is due to a self-poisoning by waste products from the muscles. As for the stiffness and tenderness of the muscles, Lagrange is inclined to attribute these to slight physical injuries of the fibres of the muscles and tendons.

A man or animal overworked becomes jaded, and this ultimate result of fatigue is discussed under the name of *surmenage.* An animal hunted to death may die from asphyxia and syncope as we have seen, but the hunted animal which escapes is not unfrequently found dead in its lair on the following day, and this death is due to an auto-intoxication by the waste products of muscular work.

Excessive muscular work produces well-recognised changes in the muscle. The sudden onset and rapid disappearance of rigor mortis in hunted animals is well known. The gourmet appreciates the tenderness and flavour of the coursed hare, and the cook knows that a coursed hare is "fit" much sooner than one shot, and that it is very liable to go putrid. Jaded muscles yield on analysis an undue proportion of extractives, and also, it is said, of alcaloidal bodies. It is very rare, except perhaps occasionally on the field of battle, that men die from acute muscular overwork, but a jaded condition, the result of "all work and no play," is not uncommon. It is asserted that the process of self-poisoning by waste products may cause a kind of typhoid fever, and Lagrange is careful to assert that it is something more than a mere predisposition to the fever, but an actual "auto-typhisation" (to use an expression of Professor Peter's), which is brought about. This requires more investigation. The disease which follows upon "forced marches," and the liability of overworked and jaded troops to succumb to various forms of ill-health, is, however, perfectly well known. Lagrange asserts that the *miasme humaine*, or androsmia, is far more marked after excessive work, and he alludes to the overpowering smell of a barrack-room which has served as a dormitory for troops after a forced march.

It is asserted also that most cases of "sunstroke" are really largely due to muscular overwork. When excessive exercise is accompanied by mal-nutrition we get a condition of wasting and chronic exhaustion, in consequence of the tissues of reserve being used for physiological combustion.

Nervous exhaustion is one of the results of muscular overwork, as it is of intellectual overwork. Lagrange states that he has seen many cases of transient melancholia occurring at the beginning of autumn, and due to the excessive labour of the harvest. As a rule the rustic is not called upon to make any great physical exertion, but from the beginning of June he often labours inordinately, working very long hours, and putting forth all his strength; his sleep is short, his diet is often unsatisfactory, and the penalty which he pays is often that of nervous exhaustion.

The ideas of J. J. Rousseau that fresh air and pure morals are a sure protection against diseases of all kinds are not tenable.

Peasant women are very liable to exhaustion, for, in addition to great physical labour, they have to endure their maternal causes of exhaustion. Nervous women are as common in the country as the town, but the manifestations of their condition are less obtrusive. They have no time for crises, but neuralgias, gastralgias, vertigoes and neuroses of every kind form the stock complaints of the female peasant exhausted by physical labour.

Fatigue results from (1) certain material changes in the organs of locomotion (slight bruising, &c.); (2) self-poisoning by waste products; (3) absorption of living tissues tending to produce wasting; and (4) dynamic exhaustion of the motor elements of nerve and muscle.

Rest has for its object the removal of the effects of fatigue, viz. (1) the cessation of the discomfort caused by the material changes in the organs of locomotion; (2) to give time for the getting rid of the waste; (3) time for repair; and (4) time for the nerve and muscle elements to gather energy.

Aptitude for exercise is one of those things that grows by what it is fed on, and, provided time be allowed for repose and the subject be well nourished, the more work a man does the easier he will do it. It is a common expression that the man in training is as "hard as nails," and this hardness of the tissues is no figure of speech, but a sober fact. As the hands get protected by a growth of epithelium, and soon cease to blister and bruise as the result of manual labour, so the deeper tissues, muscles, bones, synovial sacs, ligaments and nerve twigs soon cease to suffer from the friction of exercise.

By exercise we become disencumbered of superfluous fat, the heart and lungs perform their functions with greater power and regularity, and the nutrition of the body is improved throughout. The effectiveness and ease with which muscular acts are performed increases with practice, and, although it is not capable of demonstration, it is certain that an improvement in the nutrition of the nervous system must take place. It is certain that physical courage is to a large extent bred of physical training, and that thus one mental quality follows to some extent in the wake of bodily work.

Exercises may be divided for purposes of classification into violent, moderate and gentle; and, with regard to these, Lagrange offers a timely word of warning. The quantity of exercise taken is often difficult to measure. When a man walks upstairs, it is comparatively easy to calculate the work done in foot-tons or pounds; but, when a man is struggling with an adversary, the calculation is by no means so easy. For the same individual the best index of work done is the physiological effect produced on the lungs and heart. A violent exercise produces breathlessness before fatigue, a moderate exercise will produce local fatigue before breathlessness, and gentle exercise will scarcely produce either the one or the other. We are warned against the error of measuring the amount of work done by the difficulty of the exercise. Thus, a man who walks ten feet upstairs, and another who raises himself to the same height by a rope "hand over hand," do the same amount of work; but the muscular mass involved in the act being in the latter case relatively small, the difficulty is great, and local fatigue is liable to stop the performance. In the easy amusement of skipping, a little girl really takes a very large amount of exercise, whereas in a gymnasium she gets often a very inferior amount of exercise, and is bothered rather than amused in trying to learn some tricky *tour de force* which, when accomplished, does not produce a physiological effect in any way equal to the skipping-rope. At the present time it is the fashion to regard the trapeze as the regenerator of the human race, and we are told by gymnastic pedants that it is only by long research and profound meditation that we can use our limbs. Even in young ladies' boarding-schools it is the fashion to erect gymnastic machinery and teach difficult and grotesque exercises, notwithstanding that many a childish game produces a far greater physiological result.

Lagrange arranges his exercises in three classes:—

(1.) *Exercices de Force*, which might be called massive exercise, such as carrying a heavy load upstairs or wrestling. These exercises quickly induce fatigue and loss of breath, because the local effect on the strongly contracted muscle is severe, and the carbonic acid of which the system seeks to be rid is necessarily enormous.

These exercises demand a great expenditure of muscle, but they favour repair. They involve very little nervous excitement and very little cerebration, such as is involved in feats of skill.

The muscular contractions are powerful, slow, and sustained, and these, according to Lagrange, are very favourable for the nutrition of the muscles, because the highly oxygenated blood is long in contact with them. These exercises stimulate largely the organs of respiration and circulation, and, as they do not tax the mind or emotions, they are favourable to nutrition, and those who practise them are often herculean in form. On the other hand, in subjects who are out of training or weakly they lead to a variety of muscular mishaps and encourage emphysema and lesions of the vascular system.

(2.) *Exercices de Vitesse.*—Rapid exercises often involve an amount of work quite equal to the massive exercises, and like them they encourage the full expansion of the lungs and quicken the circulation. In a bout of fencing or a spurt of racing, the work accumulated soon equals that of the miller's man who toils up a ladder with a sack of meal, or the wrestler the whole of whose strength is antagonised, and who, in spite of supreme and continued effort, produces very little visible effect. All of these take their exercise in "big doses." Painful fatigue is not so marked in rapid as in massive exercise, because it is evident that four movements of ten kilogrammes each do not strain the muscles as does one movement of forty kilogrammes.

Rapid exercises do not make the muscles grow to the same extent as massive exercises, because, it is said, the afflux of blood to them is not so great. They cause a great development of the chest, however, and the big thorax and small calves of the professional runner are well known. In rapid exercises the response of the muscle to stimulation becomes phenomenally quick. This is partly due to the training of the muscle, but partly also to the force of the stimulation, or, in other words, to the degree of mental effort. When we "hurry," the mental effort is at its maximum.

The fatigue brought about by rapid exercise is different to that due to massive exercise, and the feeling of it is akin to that caused by moral excitement. It does not conduce to repose or sleep, and in fact seems at times to oppose them, and hence it is that nutrition after rapid exercise is less marked than after massive exercise, and those who pass their lives in hurrying work are generally thin and have smaller muscles than those who labour hard and slowly, notwithstanding that the sum of work accomplished may be the same in both cases. Competition and hurry cause great nervous excitement, and this often has the effect of putting a man or an animal off his feed. It is the racehorse or hunter which refuses its corn at night, and not the drayhorse.

Lagrange devotes a chapter to (3) *Exercices de Fond*, by which he means those gentle exercises which are practised habitually and more or less continuously. As a type of this class of exercise one may take canoeing or walking on the level. These exercises do not cause fatigue nor breathlessness, because the elimination of carbonic acid keeps pace with its formation. They do not produce a great development of the chest, because the lung is not so fully distended as in rapid exercise. These exercises are to be recommended for persons with weak lungs or heart.

Although there is no accumulation of carbonic acid, the same cannot be said of the nitrogenous waste from the muscles, which tends to accumulate, and that to a large extent, if the exercise is long continued. After a long day's shooting the man out of training feels more or less "done up" on the following day, notwithstanding that his exercise may never have produced breathlessness. It is alleged that the nitrogenous tissues in the young are very unstable, and tend unduly to accumulate during prolonged exercise, which is very trying indeed to children, whose natural aptitudes fit them for rapid exercise.

In analysing the various exercises in vogue, Lagrange points out that the tendency of the age is, in gymnastics, to put the arms to their wrong use. Trapezes, parallel bars, and rigging are the stock apparatus of the gymnasium. On these the whole weight of the body is moved by the muscles of the upper limbs, and the relatively small muscular mass and the unstable humero-scapular joint effect with difficulty what the massive legs and solid hips and pelvis do easily.

These exercises do not create the same need of breathing as do those in which the big mass of the lower limbs is involved; they are difficult and their execution is worrying, and they produce an excessive development of the muscles of the shoulders and nucha, which is in fact a deformity. It is a mistake to suppose that these exercises produce great chest development: to do this a large mass of muscle must be set in motion so as to cause the breathing to be deep. It is the runner who gets a big thorax, and not the mountebank of the trapeze, who to a moderately developed chest adds a mass of shoulder muscle which makes him look almost humpbacked.

One of the peculiarities of trapeze and bar exercises is the frequent necessity of flexing the pelvis on the trunk, a movement which is very rare in any of the habitual exercises of life. This is done mainly by the recti abdominis, which become highly developed in professional gymnasts. Trapeze and bar exercises, by increasing the firmness of the abdominal wall, prevent the growth of the belly, and, according to Lagrange, such exercises are the very best *ceinture contre l'obésité.*

The general effect of exercise is to stimulate nutrition and increase metabolism. Elimination of waste products is the first result, but in a healthy individual this waste is more than counterbalanced by quickened absorption. It therefore results that plethora is reduced while the appetite is increased. Hence we generally see, as the effect of well-regulated exercise, that the fat persons regain their figures, and the thin and ill-nourished put on flesh. The really healthy person feels a need of exercise, which should take its place along with hunger and thirst as one of the natural appetites of the body. This appetite, if not obeyed, languishes, and the lazy person becomes so encumbered with surplus tissues that movement becomes painful, and the taking of necessary exercise gets more and more difficult in proportion to our abstinence from exercise. Laziness, as we all know, is a dangerous vice which is apt to grow upon one, and the effort to throw it off becomes more and more difficult with its continuance. Lagrange combats the notion, which is very general, that the best way to "develop the chest" is to exercise those muscles part of whose function it is to elevate the ribs. It is no doubt true that well-trained and thoroughly exercised chest muscles are an advantage, and the human figure does not assume its ideal form unless the muscles of the thorax and upper limbs are well developed; but Lagrange is undoubtedly correct when he maintains that the degree of inflation of the lungs is the main factor in determining the capacity of the thorax, and, if exercise is of a kind to produce deep inspirations of the lungs, the thorax will grow big no matter what muscles are mainly in use. Inspirations are deepest when the mass of muscle in use is biggest, and, as the muscular mass of the lower limbs is greater than that of the upper, it follows that running and similar exercises are likely to cause, and in fact do cause, great development of the thorax, *i.e.* increase in size of the thoracic cage. Gymnastic machinery for the development of the chest is a mistake, and if a little patient has a narrow, retracted chest, it is best to recommend running or a skipping-rope.

The abuse of gymnastic machinery—trapeze, parallel bars, &c.—which all throw the support of the body upon the upper limbs, tends to produce an ungraceful figure and genuine deformity. The shoulder muscles are developed enormously, the upper dorsal region of the back is arched and the scapulæ tilted and dragged forward. The professional gymnast gets to look hump-backed and top-heavy. Fencing, if carried to excess, especially by young subjects, is liable to cause a slight lateral curvature of the spine towards the sword arm, and to counteract this it is advisable to use the left hand occasionally.

French boxing, it is alleged, is a far better exercise than fencing, *i.e.* for a young man. French boxing involves kicking, and thus all four limbs are abundantly exercised as well as the spinal column, which is ever moving to maintain the balance of the body, when the combatant attempts the *coup de pied* with either foot, and especially when he first of all kicks his adversary in the face with his right foot, and then delivers a second blow on the same spot with his left foot by means of the *coup de pied tournant.* Undoubtedly this French edition of the "noble art" would make the practiser as sharp and lissome as a monkey.

Among exercises which may safely be recommended, is rowing, especially with a pair of sculls, because the development of the body is perfectly symmetrical.

For the improvement of the figure and for the encouragement of an erect bearing, all exercises requiring an accurate balance of the body are recommended. It is also essential that both sides of the body should be brought equally into play. The carrying of light weights upon the head is recommended as one of the best modes of improving the figure.

It would appear that in France as in England the question of "Overpressure in Schools" has occupied the public mind, and the Academy of Medicine has passed resolutions to the effect that the scholars in elementary schools are in need of less sedentary work and more physical development. Lagrange does well to point out that there is a strong analogy between mental fatigue and muscular fatigue, and that the brain is, so to say, the organ of exercise as well as of thought, and that it is by no means always true that during physical exercise the mind is at rest.

The mental and nervous effort is often out of proportion to the visible effect. The cat prepared to spring, the fencer on the alert, watching his adversary with a sudden movement ready planned in his mind, are by no means at rest, and it is well known that fencing is an exercise which causes great nervous exhaustion, which of course is enormously increased when the foils are naked and the "play" is in earnest. In men whose nervous energies are taxed by study, fencing is not a desirable exercise, but it is excellent for those who spend a life of idleness, and who need some exercise which shall stir their minds as well as their muscles. It is, says Lagrange, an excellent exercise for those who wish to get thin or to keep thin, as the constant nervous tension helps to this end. It is noticeable that the cat has less tendency to get fat than other domestic animals, and he attributes this to the fact that it is ever on the watch, prepared to spring on any object, be it mouse, rat, fly, or what not.

Some exercises require far greater delicacy of co-ordination than others, and the perfectioning of some acrobatic movements is more a matter of co-ordinative education than of muscular development. Many a *tour de trapeze* is easy enough "when you know the way," and, although it worries the learner, the physiological result is small from the point of view of muscular exercise. For those who are working hard with their muscles exercises of the simplest kind or such as have already been fully learnt should be chosen. It is very undesirable to harass children with a drill-master or gymnast during the hours which ought to be devoted to "play." Dancing, says Lagrange, is an amusement, but "learning to dance" taxes both mind and body. He draws a graphic picture of the terrible ordeal undergone by a distinguished oculist while learning the polka. The brows knit with his mind concentrated on his legs, the feet moving regardless of rhythm, and the sweat rolling off his forehead. "Never," said he, "was I equally bothered by an operation for cataract."

While some exercises bother the mind and demand (at least at first) a great amount of attention, others, such as walking, rowing, and many childish games, are performed almost automatically. These simple exercises are those which are best for the scholar working hard for examination. Such an one ought never to be pestered with gymnastic machinery and acrobatic tricks. For the indolent person, with his intellectual faculties lying fallow, exercises which demand attention, and have in them a certain element of difficulty, are to be recommended.

G. V. Poore, M.D.

Intracranial Tumours. By Byrom Bramwell, M.D. Edinburgh: Young J. Petland, 1888.

This book is difficult to criticise, for the author has either avoided introducing much debatable matter, or has alluded to all the views concerning the subject under discussion. The result is that he has presented us with a remarkably clear and concise exposition of the principal facts known about intracranial tumours. Each symptom is explained upon the basis of physiology, by which means that dry catalogue of symptoms so common in text-books on medicine is avoided, and any senior student after reading the book would feel that he had an accurate rational knowledge of the subject. We are afraid, however, that the excellence of the book will prevent its having a very wide circulation, for cerebral tumours are not sufficiently common in general practice for the practitioners to care to obtain such an elaborate book as this is, consisting as it does of 270 pages and 116 figures, whilst the fact, that many of the more doubtful points are barely alluded to, deprives the book of the value it might have been to those who are specially interested in nervous diseases.

The author is to be congratulated on having all the way through the book clearly distinguished between those symptoms which point to the existence of a tumour, and those which indicated its exact site. This distinction is one which students frequently fail to grasp.

We are fully in accord with Dr. Bramwell in his belief that the general symptoms of cerebral tumour, such as headache, vomiting, double optic neuritis, vertigo, mental dulness, lethargy, &c., are due to the increased intracranial pressure caused either directly or indirectly by the tumour. As evidence of this pressure he adduces the separation of the cranial bones that one sees in young children in whom they are not yet firmly united. We are rather surprised that he has not mentioned the remarkable thinning of the bones of the skull seen in cases of cerebral tumour, even when the tumour does not come in direct contact with the bone. Cases have been recorded in which the thinning of the bones from this intracranial pressure was so great, that they could be pressed in like the bottom of an oil-can. The reasons why sometimes localising symptoms are fallacious are fully considered, but, as Dr. Bramwell says, the danger arising from these fallacies has been probably overestimated.

The Chapter on the causation of Double Optic Neuritis in Cerebral Tumours is a particularly clear and unbiassed account of the rival theories. The favoured view is that of Leber and Deutschmann, which is that the essential and primary cause is the presence of an irritant in the cerebro-spinal fluid, a secondary and favouring cause being the increased intracranial pressure.

The value of the work to those specially interested in nervous diseases would have been much increased if there had been a fuller discussion on the controversy about the usual and auditory centres, on aphasia, and on the rise of temperature sometimes seen in cases of cerebral tumour. The chapter on the differential diagnosis will be found extremely useful to the student of nervous diseases, for in it all the possible mistakes are considered, and by reading the chapter carefully he will learn to have them always before his mind. Attention is specially directed to the fact that tumours of the centrum ovale may produce symptoms like those of a cortical tumour, because they grow towards and irritate the cortex. This, although well known, is sometimes forgotten in cases of cerebral disease. In alluding to the greater frequency of tumours of the brain in males than in females, Dr. Bramwell adopts the usual suggestion that this may be due to the fact—the men following more dangerous occupations than women, are more liable to head injuries; but he makes no mention of a fact which must also be borne in mind, namely, that tumours of the brain are commoner in male than in female children, and one may fairly assume that the two sexes are in children equally exposed to head injuries.

The book is, if anything, too well got up, the number of figures and their size adds to the bulk of it, and presumably to the cost of it, and therefore renders it less available to students. Many of the illustrations of the pathological nature of the tumours might have been left out, for such illustrations are common enough in pathology books, and some of those showing the position of tumours might have been much diminished in size.

We can most thoroughly recommend the book to those who wish to get an accurate knowledge of the subject; so clearly is it written, that we wish there had been more of it.

HALE WHITE, M.D.

Abstracts of British and Foreign Journals.

Current Nerve Anatomy and Physiology.—Titles and Indication of Contents of recent Papers. By ALEX. HILL, M.D., Master of Downing College, Cambridge.

ANATOMY.

1. Models illustrative of Brain Growth and Cranio-Cerebral Topography. CUNNINGHAM (*Lancet*, 1888, p. 1028. *Dublin Journal of Med. Sci.*, 1888, *No.* 8, p. 151.)

2. The Topography of the Brain. J. M. CHARCOT (*Forum, New York*, v. pp. 613–626, 1888).

3. Structure of Brain. LUYS (*L'Encéphale*, vi. p. 513, vii. p. 16, viii. p. 129).

General survey of present state of knowledge.

PHYSIOLOGY.

4. New Ideas with regard to the General Physiology of the Nerve Centres, explaining the Pathogeny of Paralysis and Anæsthesia. BROWN-SÉQUARD (*Comptes Rendus Soc. de Biol., Paris*, p. 277 and p. 290, 1888.)

PHYSIOLOGY—BIRD.

5. Physiology of Bird's Brain. SCHRADER (*Pflüger's Archiv*, xliv. *Nos.* 5 and 6, pp. 175–239, pl. iii., *Nov.* 1888.)

The effects which follow removal of great brain.

COMPARATIVE ANATOMY—ANNELIDA.

6. Central Nervous System of Lumbricus. FRIEDLÄNDER *Zeit. f. wiss. Zool.*, vol. xlvii. pt. 1, pp. 47–85, pl. ix. and x., *September* 1888).

COMPARATIVE PHYSIOLOGY—FISHES.

7. The Functions of Central Nervous Systems and their Phylogeny. Part II., Fishes. STEINER (*Brunswick, Vieweg and Son*, 1888; 27 woodcuts, 1 lithograph).

The volume already published on the brain of the Frog deals with function only. Present volume considers anatomy and phylogeny also. Various functions dealt with in ten chapters:

(1) With regard to equilibration, fins removed or thrown out of function; these organs do not preside over locomotion, which depends upon pendulum-like swinging of the tail. Fishes without paired fins are never seen swimming free in water. They serve to arrest quick movement and produce backward movement. Equilibration depends on general muscle-sense. (2) Bony fishes. Skull-case opened and closed again. Bones of skull lifted from in front. Replaced. Wound closed with gelatin, which is coagulated with strong solution of tannin. In two days gelatin-cap falls off wound, by this time united (Squalius cephalus). Fore-brain removed; normal movements; able to see obstacles; appearance of volition; worm seized and swallowed one day after operation. Removal of roof of mid-brain: blindness. Removal of base of mid-brain: fish falls on back; no locomotion except on mechanical stimulation. Hind-brain cut behind lobus impar: locomotion lost; respiration ceases. (3) Amphioxus may be divided into two or three parts, each of which balances itself and moves with head-end forwards: metameric locomotor centres. (4) Scyllium catulus and canicula: fore-brain removed; locomotion as in bony fishes, but inability to ingest food; death from starvation. Cerebellum removed: no disturbance of movement on equilibration. Section above vagus origin; no locomotion. Below vagus origin: trunk makes normally balanced movements, lasting for hours, or even a whole day. (5) (6) (7) (8) Spinal cord in roach, ganoids, petromyzon, and eel. (9) "Locomotion centre" in medulla. Forced movements depend upon activity of general locomotion centre. Forced movements after one-sided injury of those parts of the brain in immediate relation to locomotion. (10) In cartilaginous fishes same results follow removal of fore-brain and of olfactory lobe: conclusion that their fore-brain is nothing else than olfactory centre. Similar phylogenetic relation of fore-brain in all vertebrates. Brain comes into existence for purpose of associating at least one higher sense organ with locomotion centre.

EMBRYOLOGY.

8. Atlas d'Embryologie. DUVAL (*Paris, Masson*, 1889, 40 plates).

EMBRYOLOGY—SPINAL GANGLIA.

9. Morphology and Development of the Nervous System of Vertebrates. BEARD (*Anatom. Anzeiger*, iii. pp. 874–898, *November* 7 and 17, 1888.)

A sketch of principal results obtained by author and published *in extenso* elsewhere. Maintains *contra* His that the "zwischen Strang" is a portion of the epiblast, which takes no share in the formation of the cranial or spinal ganglia, and that "zwischen Rinne" does not exist; on the contrary, there is in the head as in the trunk a "zwischen Strang" With regard to the essential point, the origin of the spinal and cranial ganglia for epiblastic structures outside and independent of the central nervous system. Beard and His are at one, although differing as to exact mode of growth. This origin of the ganglia and dorsal roots from metameric rudiments situate at the extreme dorsal part of the epiblast, and re-entering angle between the dorsal portion and the neural tube, is true for all sensory nerves, including the olfactory. Each typical cranial ganglion arises from two sources, the "neural ganglion" (in the situation just described) and the "lateral ganglion," developed in connection with the branchial sense organs. In frogs the lateral ganglia separate from the epiblast before the neural ganglia reach them. The neural plate in all vertebrates, amphioxus being excluded until further investigated, is a paired structure, the two halves being separated in the median line by a ciliated groove, as in developing annelids.

EMBRYOLOGY.

10. Development of the Peripheral Nervous System of Vertebrates. (Part I. Elasmobranchii and Aves.) BEARD (*Quarterly Journal Microscopical Anatomy*, cxiv. pp. 153–228, pl. xvi.–xxi., *October* 1888).

Work *in extenso* of which the abstract is given in the *Anzeiger* quoted above. Previous accounts have all, with certain partial exceptions, described the posterior roots as growing out from the primitive spinal cord in the same way as anterior roots. Beard shows by numerous sections of embryo torpedo, pristiurus, mustelus, chick, &c., that the ganglia, spinal and cranial, originate independently of the primitive spinal tube, and that the posterior roots and sensory nerve fibres grow inwards towards the spinal cord, and outwards towards the periphery from the cells of the ganglia. Details of views and points of departure from His, Froriep, and Onodi, too complicated to abstract.

EMBRYOLOGY—NEURENTERIC CANAL.

11. Neurenteric Canal and Anal Canal in Rabbit. GIACOMINI (*Archives Ital. de Biologie*, x. 2, pp. 273–294, plate, *September* 1888).

MORPHOLOGY.

12. Development of Brain Mantle in the Animal Kingdom. EDINGER (*Deutsche Med. Wochenschr.*, 29, p. 604, 1888; *Munchener Med. Wochenschr.*, 27, p. 457, 1888).

GREAT BRAIN—CONVOLUTIONS.

13. Convolution of Broca in Primates. HERVÉ (*Bull. Soc. d'Anthropologie, Paris*, xi. p. 275, 1888, plates).

14. Convolutions of Great Brain. SCHNOPFHAGEN (*Wiener Med. Presse, No.* 45, pp. 1624–5, 1888).

15. Broca's Convolution. HERVÉ (*Paris*, 1888, *Lecroisnier et Babé*, pp. 164, 10 figs., 4 plates).

Cortex—Localisation.

16. Brain of Man from whom Limb had been amputated. Arnozan (*Journal de Méd. de Bordeaux*, xvii. p. 553, 1887–8).

17. Functions of the Occipital and Temporal Lobes of the Monkey's Brain. Brown and Schäfer (*Phil. Trans. Royal Soc., London*, vol. 179, B., pp. 303–327, pl. 48–50, *August* 1888).

Extensive ablations with knife and cautery; in some cases all the parts removed at once, in other cases after successive intervals. Favourable recovery in all cases. Animals kept alive for weeks or months, then killed, brain examined: lithographs of brains.

Unilateral removal of occipital lobe: immediate and persistent blindness for all images falling upon the corresponding half of each retina. First bilateral operation: complete blindness without any recognizable changes in eye-balls; second case, blindness persistent, but not complete; lower parts of retinæ being still sensitive (small portion of external surface of right lobe was left). The angular gyri were not injured in these cases. Angular gyri removed; sensibility of conjunctiva not affected.

Temporo-sphenoidal lobes on one or both sides, partially, or completely removed. In all cases, same negative results. No sensory disturbance. In two of these cases, a remarkable disturbance of intellectual functions. Animal became for a time idiotic. Movements slow. Senses dulled. Memory very defective. Former experience lost. Slow to acquire new experience. Things, animals, and persons deliberately examined with all the senses. Animals fed with head in dish instead of picking food up. Gradual but complete recovery.

Cortex—Secretion.

18. The Influence of the Cortex upon the Secretion of Saliva. Bechterew and Mislawsky (*Neurologisches Centralblatt*, 1888, 20, pp. 353–356, *October* 15).

Dogs curarized to prevent mistakes owing to contraction of facial muscles. Secretion from submaxillary gland, when anterior part of fourth convolution, bordering sylvian fissure (corresponding roughly with face centre), is stimulated with current too weak to affect other cortical centres. More extended region for stronger current.

Cortex—Function.

19. Function of the several Layers of Ganglion Cells in the Sigmoid Gyrus of the Cat. Von Monakow (*Arch. d. Sci. phys. et nat.*, xx. 10, pp. 358–360, *October* 1888).

CORPUS STRIATUM.

20. Physiology of the Sub-Cortical Ganglia and their Relation to Epilepsy. ZIEHEN (*Deutsche Med. Wochenschr., No.* 27, p. 457, 1888, *and other journals*).

GREAT BRAIN—FORNIX.

21. Olfactory Bundle of the Cornu Ammonis. ZUCKERKANDL (*Anat. Anzeiger*, iii. 15, pp. 425–434, *June* 1888).

Foville and others have observed fibres from anterior pillar of the fornix streaming towards olfactory lobe. Structure of this part of the fornix in osmatic animals (sheep, pig, &c.), compared with that of man and other primates. In the pig the "olfactory bundle of the cornu ammonis" courses in the body of the fornix to the back of the septum pellucidum: here, like the pillars of the fornix, it bends down, but passes in front of the anterior commissure [pre-commissural fibres of Huxley], on the mesial surface of the hemisphere, towards the base of the brain, where it "streams into the temporal and olfactory lobes."

EPIPHYSIS.

22. Development of Pineal Gland. FRANCOTTE (*Archives de Biologie*, viii. 4, pp. 757–821, 1888).

CORPORA QUADRIGEMINA.

23. Optic Lobes of Teleostei and Corpora Quadrigemina of more highly organized Brains. AUERBACH (*Morphol. Jahrbuch*, xiv. 3, pp. 373–394, pl. xvi. *Nov.* 13, 1888).

CRUS CEREBRI.

24. Fibre Systems and their Degenerations in the Pes Pedunculi Cerebri. SIOLI (*Allgem. Zeitschr. f. Psychiatrie*, xl. 4, pp. 428–431, 1888).

MID-BRAIN—POST. LONG. FASC.

25. Constitution of Posterior Longitudinal Bundles (Fasciculus Longitudinalis Posterior). JAKOWENKO (*Wjestnik Psich.* 1888 : *in Russian*).

A case in which these bundles as well as a great number of other parts of the brain were degenerated. Only where the adjacent nuclei and central grey matter were also diseased could the degeneration be traced. It ascended—uninterrupted from oculo-motor nucleus to posterior commissure. These latter fibres not considered to be continuation of those farther back. Conclusion that greater number of fibres are short commissural; the long fibres probably sensory.

OPTIC CHIASM.

26. Incomplete Crossing of Fibres in Optic Chiasm of Dog. VITZOU (*Comptes rendus, Acad. des Sciences*, cvii. 12, pp. 531–533, 1888).

CEREBELLUM.

27. Ontogenesis of Cerebellum. LAHOUSSE (*Arch. de Biologie*, 1888, i. p. 43).

28. Degenerations consecutive to Extirpation, total or partial, of the Cerebellum. MARCHI (*Rivist. sper. di Freniatria*, xiii. p. 446, 1888).

Completion of previous experiments on dogs.

Conclusions: removal of one-half of cerebellum produces degeneration of medulla of same side and olive of opposite side, and to slight extent of olive of same side, and medulla of opposite side. Superior peduncles do not cross completely. Middle peduncle not true commissure, but fibres lose themselves in grey matter of pons.

One-sided extirpation also induces degeneration of direct cerebellar to certain extent of pyramidal fibres of same side. Degenerated fibres are also found in cranial nerves and anterior roots of spinal nerves, and partial fatty degeneration of musculature of same side.

CEREBELLUM—ATAXY.

29. Cerebellar Locomotor Ataxy. BORGHERINI (*Riv. sper. di Fren.* xiii. 4, 1888).

30. Case of considerable Destruction of the Vermis Cerebelli, and Discussion of so-called Cerebellar Ataxy. BECKER (*Virchow's Archiv*, cxiv. 1, pp. 173–186, pl. v., *October* 1888).

Monticulus, folium cacuminis, tuber valvulæ and pyramis of vermis replaced by two cystic tumours. Patient, eighteen years old, died of pneumonia. She never exhibited any evidence of vertigo, paralysis, disturbance of sensation, head-ache, or reeling gait.

OLIVE.

31. Minute Structure of Medullary Olive. VINCENZI (*Atti R. Acad. Med. di Roma*, 1888, pp. 171–182, 3 plates).

MEDULLA OBLONGATA.

32. Obscure but important Phenomena exhibited by a Dog after Section of one-half of the Medulla Oblongata. BROWN-SÉQUARD (*Comptes rendus Soc. Biol., Paris*, 1888, p. 407).

CORD—CENTRES.

33. Centrum ano-vesicale. ROSENTHAL (*Wiener Med. Presse*, 18, 19, 20, 1888).

Situation of this centre in man subject of considerable dispute. R. localises it in sacral cord, from observations and experiments on case in which bladder only was paralysed.

CORD.

34. Methods of investigating the Topography of the Motor Routes in Spinal Cord, &c. STEFFAHNY (*Beit. zur Anat. und Phys., Eckhard*, xii.).

SPINAL CORD—HISTOLOGY.

35. A Nucleus of Encapsuled Nerve-cells in the Antero-lateral Column of Human Lumbar Cord. CONTI (*Giorn. C. R. Accad. di Medicina, Turin*, 1888, *No.* 7).

REFLEX TIME.

36. Latent Period for Cerebral Irritation and Duration of Reflexes. NOVI and GRANDIS (*Boll. di sc. Med. di Bologna*, 1888, p. 5).

REFLEX—EYELID.

37. Centre for reflex Closure of Eyelid. NICKELL (*Pflüger's Archiv*, xlii. p. 547).

Hinder boundary of centre in the middle of ala cinerea, or perhaps somewhat higher. "Ascending" root of fifth, therefore, nothing to do with this reflex. Anterior boundary front of pons.

NERVES—CRANIAL—OPTIC.

38. The Central Ending of the Optic Nerve in Vertebrates (*Zeit. F. Wiss. Zool.* xlvii., pt. 1, pp. 1–47, pl. i.—viii., *September* 7, 1888).

Method: tissue placed in osmic acid ½% to 1%, for from 14 to 20 hours; sections cut in 70% alcohol; washed, placed in 80% alcohol 3 or 4 hours; mounted in water; ammonia run in under the cover glass. The section which is usually cut by hand very thick, becomes on the addition of the ammonia as clear as glass, the darkly-stained medullated nerves being thus rendered very easy to follow. Ammonia can also be applied to sections cut on a microtome in celloidin; they can then be mounted in balsam. Numerous illustrations of sections taken from Podarcis muralis, Lacerta viridis, Trapidonotus natrix, Emys Europæa, Testudo græca, Rana esculenta, Bufo vulgaris, Triton cristatus, Siredon pisciformis, Carassius auratus, Macropodus viride-auratus, Anguilla vulgaris, Lepadogaster Gouanii, sparrow, Parus major, hen, dove; Mus musculus, Mus decumanus albus, Cavia cobaya, Erinaceus Europæus, Lepus cuniculus.

General results: fibres followed to their real ending in the "corpora opticum" (tectum opticum and corpora quadrigemina anteriora). In some types optic fibres pass through and cross over in the tuber, but return to optic tract. They are mixed with fibres of (1) commissura inferior; (2) fibræ ansulatæ; (3) decussatio inferior of birds (perhaps part of system 2); (4) numerous other thalamus fibres which make complicated plexus in mammals; (5) peduncular fibres which traverse the tract in neighbourhood of chiasma; (6) fibres traverse corpus geniculatum thalamicum, but do not end there; numerous other thalamic and cerebral fibres are mixed up with optic fibres in this region. Various strata of zonal fibres have to be distinguished. Object of paper to distinguish the primitive and proper optic fibre from those various tracts which take advantage of same routes.

II. Morphology of corpus opticum. Grows from upper wall of middle cerebral vesicle. Corpus posterius (quadr. post.) belongs rather to hind-brain. Originates in lateral intermediate region between mid- and hind-brain. The posterior tubercles of corpora quadrigemina of mammals are derivatives of corpus posterius of lower vertebrates.

NERVE—CRANIAL—AUDITORY.

39. Striæ Acusticæ in Man. VIRCHOW (*Arch. f. Anat. u. Phys.* 1888, 3 and 4, p. 392).

Demonstration by means of transverse sections of medulla that in early life the stria acustica forms a strong bundle recognizable with the naked eye, running from the auditory nerve to the middle line, where it plunges into the raphe. It causes a local enlargement of the raphe, in which it seems to run far ventralwards.

40. Conductor Sonorus (*Bergmann*), and Origin of Acoustic Nerve. NUSSBAUM (*Med. Jahrbuch*, 1888, 5, p. 187–195).

41. Conductor Sonorus, and Remarks on Origin of Auditory Nerve. NUSSBAUM (*Med. Jahrbücher*, 1888; also *Neurol. Centralbl.* 1888, 24, p. 673, *December* 15).

The bundle which ascends from the striæ acusticæ received above name, with German synonym Klangstab, from Bergmann. By Stilling it was regarded as inconstant root of trigeminus. N. cannot decide between these two connections; describes bundle as surrounding in its course a round group of ganglion cells, originating in tegment, crossing to opposite side in dorsal part of raphe, and passing partly to floor of fourth ventricle, partly to locus cæruleus.

NERVES—CRANIAL—VAGUS.

42. Central Ending of Vagus and Connection with Fasciculus Solitarius of Lenhossek. BECHTEREW (*Wjestnik Psichiatrii*, 1888, 2: *in Russian*).

43. Anatomy and Physiology of Vagus Nerve. DEES (*Archiv f. Psychiatrie*, xx. 1, pp. 89–102, pl. 1, *October*, 1888).

Literature of the several origins assigned to vagus.

Litter of new-born rabbits: vagus exposed in neck, cut and peripheral portion torn out. Animals killed at fifth week. Vagus to next branch above section normal thickness, but greyish red, and evidently principally connective tissue. Sections of medulla stained in aniline-black. Atrophy on same side of nerve-cell of (1) dorsal vago-glossopharyngeal nucleus, (2) ventral vago-glossopharyngeal nucleus. Shrinking of solitary fasciculus. Cells of ventro-medial portion of dorsal nucleus completely destroyed. Concluded from this, that axis-cylinder processes of these cells go to motor nerves of same side, for thorax; that N. intermedius Wrisbergi does not arise from proximal portion of the dorsal vago-glossopharyngeal nucleus. Ventral vago-glossopharyngeal nucleus lies between olive and lateral column. Well-defined group of cells; continuation caudalwards of facial nucleus, although separated by nerve tissue. Bundle goes directly backwards to solitary fasciculus. "Nervus accessorius vagi is not the motor laryngeal nerve." The ventral vago-glossopharyngeal nucleus is the nearest centre for the innervation of the muscles of the larynx. Fasciculus solitarius is the ascending sensory root of the N. vagus et glossopharyngeus. No evidence with regard to origin *viâ* the raphe. No origin of vagus fibres from funiculus teres.

44. Vagus Extirpation. DEES (*Neurologisches Centralblatt* 1888, 21, p. 596, *November* 1).

Note to paper by author in *Arch. f. Psychiatrie*, xx. 1.

NERVES—PERIPHERAL.

45. Nerve Supply of Mouth and Nasal Fossæ in Vertebrates. GAUPP (*Morphol. Jahrbuch*, xiv. 3, pp. 436–490, pl. xix. *November* 1888).

NERVES—PERIPHERAL—SYMPATHETIC.

46. Sympathetic System of Fishes. CHEVREL (*Comptes rendus, Acad. des Sciences*, cvii. 12, pp. 530–531).

47. Some Points in the Physiology of Gland Nerves. BRADFORD (*Journal of Physiology*, ix. 4, pp. 287–316, *November* 1888).

HISTOGENY.

48. Histology of Fœtal Brain. MAGINI (*Atti Accad. dei Lincei, rendiconti*, iv. 12, pp. 760–764, 1888, plates).

49. Genesis of specific-Nerve cells and the intimate Structure of the Central Nervous System of Birds. FALZACAPPI (*Bollett. Soc. di Naturalisti in Napoli*, ii. 2, pp. 185–193, 1888).

HISTOGENY—CORTEX.

50. Development of Elements of Cortical Layers of Cerebrum and Cerebellum. VIGNAL (*Arch. de Physiologie*, xx. 7, pp. 228–255, pl. v. and vi., *October* 1888).

Method of hardening; early fœtal brains; equal parts alcohol and osmic acid; also Flemming's and Henneguy's methods. Second half fœtal life: *alcool au tiers* (32° centésimaux) twenty-four hours; picro-carminate of ammonia (1 per cent.) two or three days; osmic acid, twelve to twenty-four hours. Literature. Brains of rabbits; brains of human fœtus, five and a half months; two sorts of nuclei colouring to different depths, but otherwise similar. Darker nuclei found of larger size; belong to pyramidal cells. Seven months: three layers; (1) 250 μ thick; (2) 1237 μ cells in series vertical to surface. At the junction of these two layers are found, the developing large pyramids with all the characters in miniature of the adult elements, homogeneous protoplasm staining deeply in osmic acid, irregular, giving off processes; large, round, or oval, clear, dark-bordered, granular nucleolated nuclei. In matrix surrounding these cells, smaller nerve nuclei and neuroglia nuclei. These cells at the seventh month correspond to the large cells of the spinal cord at four months.

51. Ditto. *Continuation* (No. 8, pl. ix.–xii., *November* 15, 1888).

Adult neuroglia cells of cortex resemble neuroglia cells of the cord in the fifth month of intra-uterine life. They do not become distinct until end of sixth month. Seventh month easily freed from surrounding matrix; they possess a spherical or ellipsoidal dark-bordered nucleus, staining pale rose in picro-carmine. After this, neuroglia cells difficult to dissociate from matrix of cerebellum. In early periods, the superficial layer is full of closely packed nuclei, belonging, as the author believes, to migratory cells. Clear layer to be found between this layer and "granular layer," in which Purkinje's cells make their appearance at sixth month. The numerous hæmatoxylin-staining nuclei V. regards as belonging to neuroglia; rarer larger eosin-staining nuclei as nervous.

HISTOGENY—MEDULLA.

52. Development of Medulla Oblongata. HIS (*Archives des Sciences physiques et naturelles*, xx. 10, p. 356, *October* 1888).

HISTOLOGY—NEUROGLEIA.

53. Interstitial Tissues of Central Nervous System and Cerebro-spinal Nerves. PETRONE (*Gaz. degli Ospitale, Milan*, 1888, *Nos.* 4 and 11).

HISTOLOGY—NERVE ENDINGS—TENDON.

54. Termination of Nerves in Tendons in Vertebrates. PANISINI (*Riforma Medica, Rome*, iv. p. 752, 1888).

Trzebinski on the Action of various Hardening Methods upon Ganglion Cells. (*Arch. für. Path. Anat. u. Physiol. u. für. Klin. Med.* Bd. cvii. H. 1.)—The occurrence of feeble staining, vacuolation, clouding and granular disintegration in the ganglion cells of the spinal cord of dogs and rabbits that had been poisoned with phosphorus or arsenic was described by Popow and Danilo as being the direct result of that poisoning. More recently Kreyssig has observed exactly similar conditions in the spinal ganglion cells of healthy animals as well as in animals subjected to the lethal action of those substances.

Finding those specified changes were absent in specimens that were examined by the fresh method, Kreyssig was led to attribute them to the media used for hardening the tissue.

Professor Flesch has in great measure confirmed Kreyssig's observations. Nissl still later has declared that the ganglion cells are altered by Müller's fluid, but are unaffected by alcohol.

The author has investigated the question by systematic experiments, using the spinal cord of healthy dogs and rabbits, and of a rabbit which had been submitted to phosphorous poisoning for eight days.

The methods of hardening were:—

1. In Muller's fluid four or five weeks: the tissue was then either washed and transferred to absolute alcohol (or 10 per cent. alcohol for five days, then absolute), or it was put into alcohol, and kept dark, without previous washing.
2. In alcohol: absolute from the commencement, or weak at first and gradually increasing the strength.
3. In chromic acid: the first six hours in 0·1 per cent., then in 0·25 per cent., for forty-eight hours, subsequently in alcohol, or a mixture of alcohol and Muller's fluid.
4. In 10 per cent. mercuric perchloride solution for eight days, afterwards in alcohol containing 0·5 per cent. of iodine.

Borax carmine, alum carmine, Weigert's hæmatoxylin, and magenta red were the principal stains employed after the chemical treatment: the fresh preparations were coloured with methyl green.

All the last mentioned (dissociated elements and frozen tissue sections) gave results corresponding with those of Kreyssig: the ganglion cells on the whole showed no marked difference in the intensity of their staining; they had no pericellular spaces, and such details of structure as granules and striæ or fibrillation could be recognised. On the other hand, in all the hardened preparations, the ganglion cells manifested striking changes in the form of pericellular spaces, vacuolation, or other alterations of the cell substance, both with respect to its intimate structure and its receptivity for various colouring agents.

The pericellular spaces were largest and most numerous in the specimens which had been hardened in alcohol alone. They were larger and more irregular in the spinal cord of dogs than in that of rabbits. Sections from different parts of the same cord, after identical preparatory treatment, presented great diversity with regard to the number and size of the spaces: even in the same section the individual ganglion cells were unequally affected. Numerous and extensive pericellular spaces would be found in one anterior horn, while in the opposite corner they were sparse and ill-defined. Sometimes they were large in the cervical and lumbar regions, scarcely perceptible in the dorsal. They in general were less marked in connection with the pale tinted cells than around the deeply stained.

The bridging over of the pericellular space by extremely delicate filaments from the shrunken protoplasm, as described by Flesch, was often seen, more especially in Müller's fluid or chromic-acid preparations.

Vacuoles were observed, in small number, in specimens hardened either in chromic acid, Müller's fluid or alcohol—most abundant in the first named. None existed in the fresh tissue, nor in that hardened by corrosive sublimate. No vacuoles were discovered in the nuclei of any of the preparations.

1. In preparations hardened after Muller's method, or any modification thereof, the cell protoplasm was indifferentiated, homogeneous, and possessed, when stained with borax carmine, a peculiar vitreous lustre, calling to mind the appearance of hyaline or amyloid tissue.

After due allowance for obvious sources of error in estimating the degree of tinting, there remained a considerable number of ganglion cells which resisted, more or less completely, all the staining reagents used by the author.

The contour and minute structure of the nuclei came out clearly when not obscured by over-staining. The degree of their coloration bore no constant relation to that of the cell substance.

2. When hardened in alcohol, the cells were sharply contoured: granulation or striation of their substance could generally be recognised. In the latter case the striæ—deeper tinted than the

clear ground substance—usually coursed from the periphery towards the nucleus, leaving, however, a free zone around it.

The differentiation of the cell structure was clearly brought out by alum carmine or magenta red, and was analogous to that noted in the fresh tissue. Large pale ganglion cells were rarely found; the protoplasm of those existing appeared coarsely granular. The nuclei tended to be more distinct than those of the preceding category: they usually were less deeply stained than the body of the cell, and allowed one or more darkly tinted nucleoli to be perceived.

3. The nuclei of the ganglion cells in chromic-acid specimens were very clear in outline and minute features. On the other hand, the cell bodies themselves were obviously more changed in these preparations than in all others. This was shown by the large pericellular spaces, the coarse granular differentiation of the cell protoplasm, and the relatively frequent presence of marginal and genuine vacuoles. Partial or complete disintegration of some of the ganglion cells, indistinctness of their contour, and clouding of their substance, also were more commonly seen than in other preparations—some nuclei being surrounded by granular detritus, others lying free.

Pale cells were less numerous than in category 1.

4. The corrosive sublimate process gave results similar in many respects to alcohol preparations, but the shrinking of the cell body—thus also the pericellular space—was less. Structural details were plainly defined. Pale cells and vacuoles were absent.

Briefly summarised, the author concludes that certain chemical hardening media produce variations in the staining capacity of the ganglion cells which are not apparent in the cells of the fresh cord—Müller's fluid acting with special potency in this way. Whether the effect in reality constitutes a micro-chemical test, of far-reaching import in connection with the anatomical and physiological relationships of the affected cells, is yet an open question.

As neither pericellular spaces nor vacuoles were found in fresh tissue, it may be assumed that their presence in certain of the hardened specimens was due to the chemicals used.

The changes caused by alcohol are less disturbing and not so liable to lead to erroneous conclusions as those produced by chromic acid or its salts. They are in no way more pronounced when absolute alcohol is used from the commencement than when a weak mixture is first employed.

The examination of the spinal cord of the animal poisoned by phosphorus gave entirely negative results, so far as any specific histological change is concerned.

Heusser on a Case of Lymphosarcoma of the Hypophysis Cerebri. (*Ibid.* Bd. cx. H. 1.)—The patient, a female, aged 64, came under treatment for cancer of the rectum. Four weeks prior to admission left ptosis commenced; two weeks later it had become complete. Concurrently with this palsy, violent pains in left side of face, radiating to the vertex, set in, and visual power in the left eye became considerably impaired. The headache, dizziness, and nausea, which had troubled patient for some time, were attributed to the great exhaustion induced by the rectal disease. A palliative operation in connection with the latter ended in fatal collapse.

A reddish tumour of moderately firm consistence, about the size of a prune, was found occupying the sella turcica, the left cavernous sinus and optic foramen, reaching also into the left ectemoidal cells. The tumour was of irregular shape: its base was traversed diagonally by the left optic nerve. Near to the left of the nerve was the ophthalmic vein with pretty wide lumen. The chief mass of the tumour was external to this vein. The base of the tumour surrounded the internal carotid artery, the lumen of the latter being scarcely diminished. The left third, fourth, and sixth nerves were also embedded in the growth, the first mentioned being compressed into a quadrangular shape.

The tumour was mainly formed by uninuclear round cells of nearly uniform size. The nuclei were round, or here and there oval, and very rich in chromatin. Each nucleus almost filled its cell, the protoplasm of the latter being very scanty. Around the vessels, lying amongst these lymphatic elements, were small round or spindle-shaped cells which stained more deeply than the former, and were regarded as granulation tissue cells. The cell masses, which in many places had a follicular arrangement, were surrounded by a delicate and extremely vascular reticulum. In all parts of the tumour numerous red blood-corpuscles filled the small vessels or lay scattered free in the tumour mass. None of the normal structure of the hypophysis was visible.

The sheath of the optic nerve was invaded by the neoplastic cells. Around and within this sheath there also were inflammatory elements, migrated leucocytes, disseminated through the whole nerve, but in greatest number at the periphery.

Besides masses of red blood-corpuscles, very numerous round cells lay within the internal carotid and the ophthalmic vein. The intima of the former was here and there displaced by subjacent accumulations of those cells: at one part, a thrombus partly occluded the vessel.

The presence of osseous lamellæ in the midst of the neoplasm—entirely surrounded by it—pointed to erosion and exfoliation caused by the proliferous tumour elements.

In addition to the inflamed condition of the optic nerve, Weigert's method showed marked degeneration of its peripheral fibres: at some points the latter had almost disappeared, being replaced by connective tissue. Corpora amylacea were found in the nerve, especially at the periphery.

Lymphatic cells were found in the nerve sheaths of the corresponding third, fourth, and sixth nerves, more particularly where the nerves were compressed by the tumour. A few corpora amylacea could be seen in these nerves, no other degenerative change. E. Birt.

Eppinger on Miliary Aneurisms of the Cerebral Vessels (*Arch. path. Anat.* Bd. cxi. Hft. 3).—This paper is a reply made by the author to critiques (respectively by Zenker and Lowenfeld) on the appendix to his recently published work on the Histogenesis and Etiology of Aneurisms. In these he was charged with an unauthorised attack on the views of Charcot, Bouchard, and Zenker regarding miliary aneurisms and their relation to spontaneous cerebral hæmorrhage. Professor Eppinger pleads in justification that he has been misconstrued by his reviewers in regard to a point on which he has laid great stress throughout his entire work, viz. his exposition of the nature of an aneurism, and which is one that does not allow him to accept that described by Charcot, etc., as such according to his definition. Extended research compelled the author to emphasize the need of a greater restriction as to the meaning of aneurism. His diagnosis of the true aneurism comprised essentially, the interruption of the elastic coat at the entrance to the swelling, its absence from the wall of the same, and the continuous transit of the remaining vessel-wall, entire or isolated, leading to or from the aneurism. Hence he rejects simple ectasie, in which, changed or unchanged, the laminæ are continued over the swelling, dissecting and spurious aneurisms. Being convinced that the changes, a part of aneurism as accepted by him, are accomplished in a three-fold manner, he sets forth three forms of such conditions: (*a*) congenital, (*b*) parasitical (including mykotische-embolische, equi-verminosum, and arrosious aneurysma occurring in pulmonary tuberculosis), and (*c*) simple, traumatic. In these, he has been able to comprehend all known aneurisms possessing the required characters. He insists especially on his definition of aneurism as representing a fixed and, histologically, well-marked disease of the artery; and whilst perfectly recognising the miliary aneurisms of Charcot and Bouchard, he has excluded them from his category as not conforming to such definition, discarding both them, as simple ectasies and perivascular changes. Atheroma as a basis of moment for the production of aneurism he also discountenances. Finally is pleaded that, not until the standpoint assumed by the author, regarding the fixed nature of aneurism, can be rendered untenable by indubitable examinations and pathologico-anatomical inferences from the same, should such harsh criticisms as have been suffered by him be justifiable. There is much interesting matter in Professor Eppinger's paper, but unless viewed in conjunction with his original work, not to hand, it would be of little service to produce it. F. St. John Bullen.

Haig on Headaches, Fits, and Mental Depression due to the Abnormal Uric-Acid Elimination.—Dr. Haig has pointed out (*St. Bartholomew's Hospital Reports*, vol. xxiii. p. 210) the close clinical relationship of epilepsy to a form of headache, which he identifies with migraine, and believes to be due to the uric acid in the blood; and has shown (*Neurologisches Centralblatt*, March 1st) that the fits in some cases of epilepsy are accompanied by an excessive excretion of uric acid exactly resembling that which occurs in the uric-acid headache. See also papers on the excretion of uric acid under various drugs (*Medico-Chirurgical Transactions*, vol. lxxi.) and Thesis for the degree of M.D., Oxford (abstract in *British Medical Journal*, vol. ii. pp. 10, 88). Dr. Haig believes that such cases of epilepsy may be successfully treated by diet or drugs which promote the elimination and prevent the retention of uric acid in the body.

In several of these papers he has mentioned some interesting symptoms which serve, he thinks, to link headache, epilepsy and mental depression to each other, and also to gout.

He has also pointed out in the above Thesis, and more recently (*Practitioner*, November 1888), that mental depression is often due to excess of uric acid in the blood, and that it may be controlled and successfully treated by acting on the uric factor

That there is an intimate connection between uric acid excretion and this mental condition is shown by, *inter alia*, the fact that when a plus excretion of uric acid is going on under the influence of an alkali, there is depression, bad temper, and lessened mental activity. On the other hand, when there is a minus excretion of uric acid under the influence of an acid, there is a bright and cheerful feeling with increased mental activity; excessive excretion meaning, the author believes, excess in the blood, and *vice versâ*.

Many people suffer from some amount of mental depression during the "alkaline tide" of the morning, when the excretion of uric acid is normally at its highest point; and mental depression may be caused in many people by giving alkali to produce an artificial alkaline tide.

The fits in some cases of epilepsy are preceded by a minus excretion (or retention) of uric acid, and are accompanied by a plus excretion of uric acid; and it has been observed that some patients are able to foretell the advent of their fits by the bright and joyous feelings which correspond, as there is no doubt, to the retention preceding them. A precisely similar fluctuation in mental conditions has been noticed by Haig and others in the case of the uric-acid headache, no doubt having a precisely similar causation.

An attack of melancholia may terminate suddenly in an attack of gout, and if melancholia is due, as Haig believes, to uric acid in the blood, a dose of acid in driving it out of the blood into the joints will cure the mental and produce the bodily disease in a way that it is very easy to imitate; for any one who is suffering from a uric-acid headache can cause pricking pains in his joints, more or less severe, by driving it out of his blood with an acid; and the same dose of acid, if given to a gouty patient, will at once make the joint pains worse.

Further, the author considers it to be a very important distinguishing mark of the mental depression due to uric acid, that in those who have joint pains it does not occur along with the pains, but alternates with them; thus a gouty patient says: "I had gout two or three years ago; now the pains have gone, and I have depression and heaviness worst in the morning." He is in poor circumstances, or otherwise run down; if you can improve his diet and nutrition, and so raise his acidity, you will bring back his gout and remove his mental depression.

Many patients who suffer from uric-acid headache suffer also from some mental depression, which can be cured in the same way as the headache, by a dose of uric acid; their place being taken by bright and happy feelings of well-being.

Dr. Broadbent has pointed out that a high-tension pulse is a very constant symptom in cases of melancholia; and Haig has remarked that the pulse tension during the uric-acid headache is generally very high, and that, *cæteris paribus*, pulse tension varies with the amount of uric acid present in the circulation, so that it is possible, by washing an excess of uric acid into the blood with an alkali, to produce headache, or mental depression and increased tension of pulse, the excretion of uric acid in the urine rising *pari passu* with its increase in the blood; or if these conditions are present, the headache or depression may be driven away, the pulse tension reduced almost to nothing, and the excretion of uric acid in the urine diminished by giving an acid. This fact seems to prove that the mental depression, and the high-pulse tension which accompanies it, are both due to the excess of uric acid in the circulation.

The treatment of such conditions is that of gout and of the uric-acid headache: a diet poor in nitrogen; or drugs, such as salicylate of soda, which eliminate uric acid. This drug, when given for gout or epilepsy, often has a marked effect in improving the spirits; and it differs in this important respect from an alkali; that while causing plus excretion of uric acid, it yet causes no headache or mental depression.

T. W. Shore on the Morphology of the Vagus Nerve.—(*Journal of Anatomy and Physiology*, vol. ii., part 3, April 1888.) The Author prefaces his remarks on the morphology of the vagus, by summarizing the leading features of its anatomy and development, in petromyzon, elasmobranch fishes, and rana, together with its development in the chick. He remarks that if Gaskell's researches, on the visceral nerves of the dog, hold good for lower types, a perfect spinal segmental nerve must contain somatic motor, somatic sensory, and splanchnic (motor and sensory) fibres, and that inasmuch as the vagus in amniota (with the exception of the small dorsal branch), as well as in fishes (excepting the lateralis and the afore-mentioned small dorsal branch), is wholly visceral in distribution, the current hypothesis that it (the vagus) is a compound of a variable number of *segmental* nerves is untenable. He regards the visceral portion as representative of the coalesced visceral rami, corresponding to the anterior end of the alimentary tract, and including the branchial nerves of fishes. He supports

Hubrecht in identifying the ganglia of the vagus, and other cranial *nerves*, with the brain lobes, of the nemertea, and further thinks that they probably also represent the cerebral ganglia of annelida and arthropoda. The ramus lateralis he regards with Hubrecht to be identical with the lateral nerve strands of nemertea, and, in addition, regards it as being homologous with the main nervous system of annelida and arthropoda, and possibly also with the cœlenterate nerve ring. The study of the vagus may, in his opinion, throw light on the question of the chordate ancestor, but will not help to any great extent in solving the problem of the segmentation of the vertebrate head. In conclusion, he suggests that the following points should be elucidated on the plan initiated by Gaskell on the dog, *i.e.* by studying the histological composition of the various parts of the vagus.

1. Does the "ramus visceralis vagi" resemble in structure the visceral portions of spinal nerves?
2. Are the fibres of the branchial nerves of the same nature as those composing the "ramus visceralis"?
3. Do the fibres of the "ramus lateralis" resemble histologically those of the rest of the nervous system?
4. Do the spinal metameric nerves of the segments corresponding to vagus visceral distribution, themselves contain visceral fibres?

With these questions he hopes to deal in a subsequent paper, to which this one must be regarded as a preface.

W. L. SYMES.

Ball on Traumatic Aphasia relieved by the Removal of a Blood-clot from the Cerebrum (*Dublin Journal of Medical Science*, September, 1888).—The author records a case of peculiar interest successfully operated on by him for the cure of aphasia. The patient had been struck a blow in the head with a penknife ten days before admission to the hospital. He had noticed, since the blow, increasing difficulty in using the right words. On examination, a small scab was found adherent to the scalp, over the squamous portion of the left temporal bone. This, when detached, showed a cicatrix extending deeply through the temporal muscle. There was well-marked motor aphasia, word-blindness, and word-deafness. No paralysis could be detected. Five days after admission, as the symptoms had increased, an operation was determined on. A flap was turned down including the cicatrix, and a wound of the squamous portion of the temporal bone, such as could be caused by the small blade of a penknife, was found. A circle of bone was removed, containing in its centre the wound in the bone. The knife was found to have penetrated the dura mater and brain. The dural wound was enlarged and a Sims' forceps was passed into the brain wound, and was gradually separated. A dark-coloured blood appeared and was extruded by the internal brain pressure; more clot was removed by Sims' forceps and weak perchloride irrigation, a drainage tube was introduced, and the external wound was sutured. On the evening of the same day the patient carried on a long conversation with very few mistakes in his selection of words. The following morning aphasia was again increased, but disappeared on cleaning the blocked drainage tube. The recovery was, after this, uninterrupted and complete.

J. W. White, M.D.

J. Hoffmann on Tetany (*Deutsch. Arch.*, 1888, xliii. 53).—This paper is based on eleven cases of tetany, reported in full, and accompanied by numerous references to cases of other observers and their opinions concerning the disease. In all but one of the author's patients the affection began between the ages of sixteen and twenty-five years. He is disposed to believe that the nature of the occupation is not without influence on its etiology, and that shoemakers, seamstresses, clerks, &c., are, perhaps, especially liable to it. After reviewing at some length the literature relating to the bearing of the thyroid gland on tetany, he concludes that, in all probability, the removal of this organ is one of the etiological factors. He does not agree with Strümpell that they are endemic influences in its production. The spasm was rather widely spread in the cases which he reports, and was always bilateral. The attacks lasted from several minutes to some hours, and in one instance ten days. The premonitory fibrillar muscular trembling was absent in most cases; the tendon reflexes were usually normal, but in one case diminished, and in another nearly absent. The attacks of spasm were always preceded or accompanied by paræsthesias. Trousseau's symptom (the production of an attack by compression of the main artery of a limb) was always present during the height of the disease, while the compression of a nerve had this effect in but one case. The disappearance of this symptom is in no sense a sign that the disease has terminated, but only that it is in abeyance. The galvanic and faradic electrical excitability of the nerves was much increased in all cases in which it was carefully examined; and even the facial and the hypoglossal nerves may share this excitability. An increase of the mechanical excitability of the motor nerves was absent in but one instance. A series of experiments convinced the author that the mechanical and electrical excitability of the sensory nerves is increased. The disease lasted in the author's cases from half a year to twenty-one years. Most commonly the attacks occur in groups, lasting some weeks, and with variable periods between them, in which there exist only certain symptoms of latency; or the intervals may continue for years, with absolutely no sign of the affection.

According to most authorities, the prognosis is favourable, though the disease may last for years. Death occurs only exceptionally, from exhaustion or spasm of the diaphragm. Certain of the author's cases exhibited evidences of paralysis, for which the tetany was apparently responsible. Frequent shedding of the finger-nails and a brown pigmentation of the skin of the hands and face was observed in one instance, and the author considers the probable cause of the latter to be nervous influence combined with repeated small extravasations of blood into the skin, induced by the muscular spasm. Hoffmann reports the results of autopsies which have been made, and quotes extensively the opinions of various writers concerning the pathological anatomy of tetany; all of which indicates that, though fine molecular nutritive changes of the nervous system are almost universally considered to be the cause of the disease, making it, therefore, a neurosis, the seat of the changes is still a much disputed point. He obtained the best therapeutical results from bromide of potash, morphia, and the galvanic current. In an appendix the author reports four additional cases of the affection, in two of which shedding of the nails took place, accompanied, in one instance, by falling out of the hair. He shows still further the positive influence of extirpation of the thyroid gland on the production of tetany, and calls attention to the fact that attacks seldom occur in summer, cold seeming to be a strongly exciting factor.

Oppenheimer on Railway Spina (*Dublin Journal of Medical Science*, October 1888).—Certain symptoms are common to all, or at least to the majority of cases of nervous disease consequent on spinal concussion without external injury. These symptoms are chiefly psychic, and belong to the affection, mental depression and irritability occupying the foreground. The mental depression is accompanied by anxiety of mind, the patient's thoughts being always occupied, even in his dreams, with the accident, but is distinguished from pure melancholia both by the abnormal irritability always present, even in persons previously remarkable for their phlegmatic indifference to internal excitations, and by its hypochondriacal character. Severe intellectual disturbance is rare, but the author has seen a considerable degree of dementia with weakness of memory, also cases of hallucination, and even of traumatic insanity, requiring seclusion. Giddiness and cramps, varying between "petit mal" and true epilepsy, are not infrequent; but care must be exercised in diagnosticating such from purely hysterical attacks.

In the domain of the special senses, a mixture of hyperæsthesia and anæsthesia, or rather their juxtaposition, is the most frequent characteristic—*e. g.* hyperæsthetic zones are found in the anæsthetic cutaneous areas; acuteness of vision is lowered, while the eye is extremely sensitive to light; the auditive faculty is lessened on the whole, while certain sounds react abnormally. Sensory anæsthesia is a very important subject, and observations have been made in Westphal's clinic with a view to distinguish genuine from simulated symptoms. The anæsthesia, *e.g.* of an extremity, does not usually follow the course of the nerve implicated, but is spread over neighbouring nerves, and is often overlooked. Typical girdle-feeling, and a corresponding anæsthetic zone, are rare. Pains are frequent, and usually of the dull kind, especially headaches. Reflex excitability is more often lessened than increased. The tendons always react. Motility is almost always affected; the patients move about slowly, and without energy; the spine is maintained as fixed as possible, sometimes reflex muscular symptoms hinder locomotion. The various ways of walking are remarkable, and may excite suspicion of simulation. There is no doubt that the usual signs here do not, as a rule, correspond with those dependent on "material diseases" of the central nervous system. When one foot drags, it is not carried as in hemiplegia. Tremor is frequent, but it resembles that of hysteria far more than that of sclerosis. Swaying on closure of the eyes is very frequent. As to the motor cerebral nerves, the speech suffers most, though there is no trace of aphasia. Speech is irregular and interrupted.

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